

ST. FRANCIS HOSPITAL ISSUE

# DELAWARE STATE MEDICAL JOURNAL

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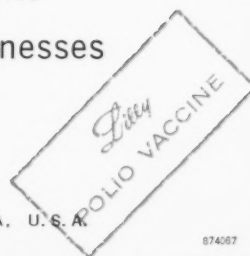
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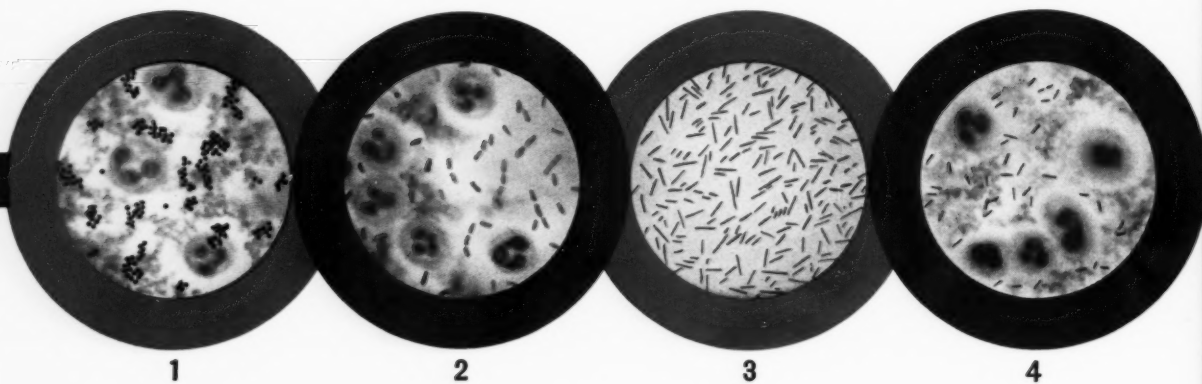
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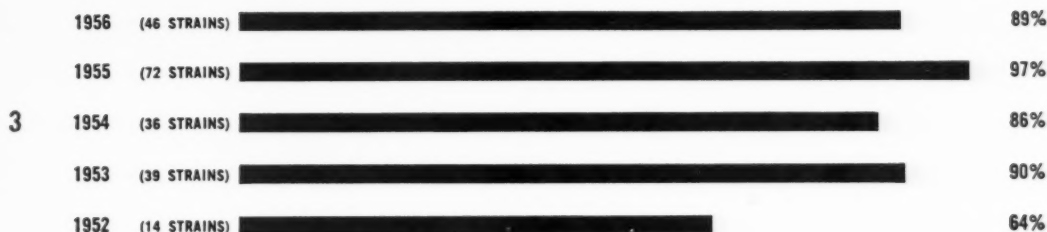
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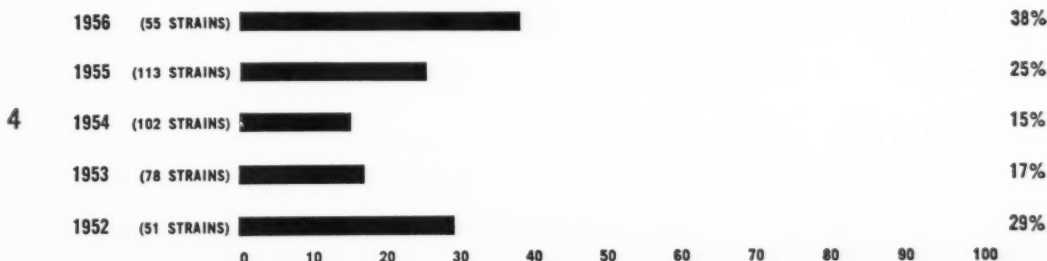
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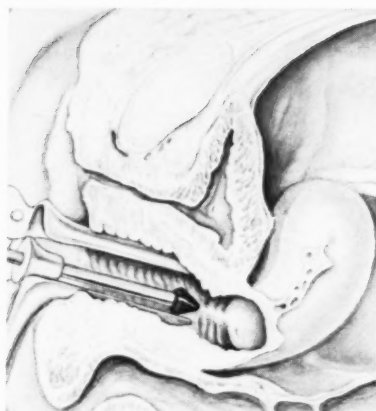
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Birth	10	10	2	3	6	320
1	12	13	2½	4	6	390
2	15	13	3	4½	6	480
3	17	9	3	5	5	520
4	20	11	3½	6	5	610
5	23	11	4	6½	5	700
6	26	10	4	7	5	760

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Age Months	Evap. Milk Fluid Oz.	Water Oz.	KARO Tbsp.	Each Feeding Oz.	Feedings in 24 Hrs.	Total Calories
Birth	6	12	2	3	6	380
1	8	16	3	4	6	532
2	9	14	3	4½	5	576
3	10	15	3½	5	5	650
4	12	18	4	6	5	768
5	12	21	4	6½	5	768
6	13	22	4	7	5	768

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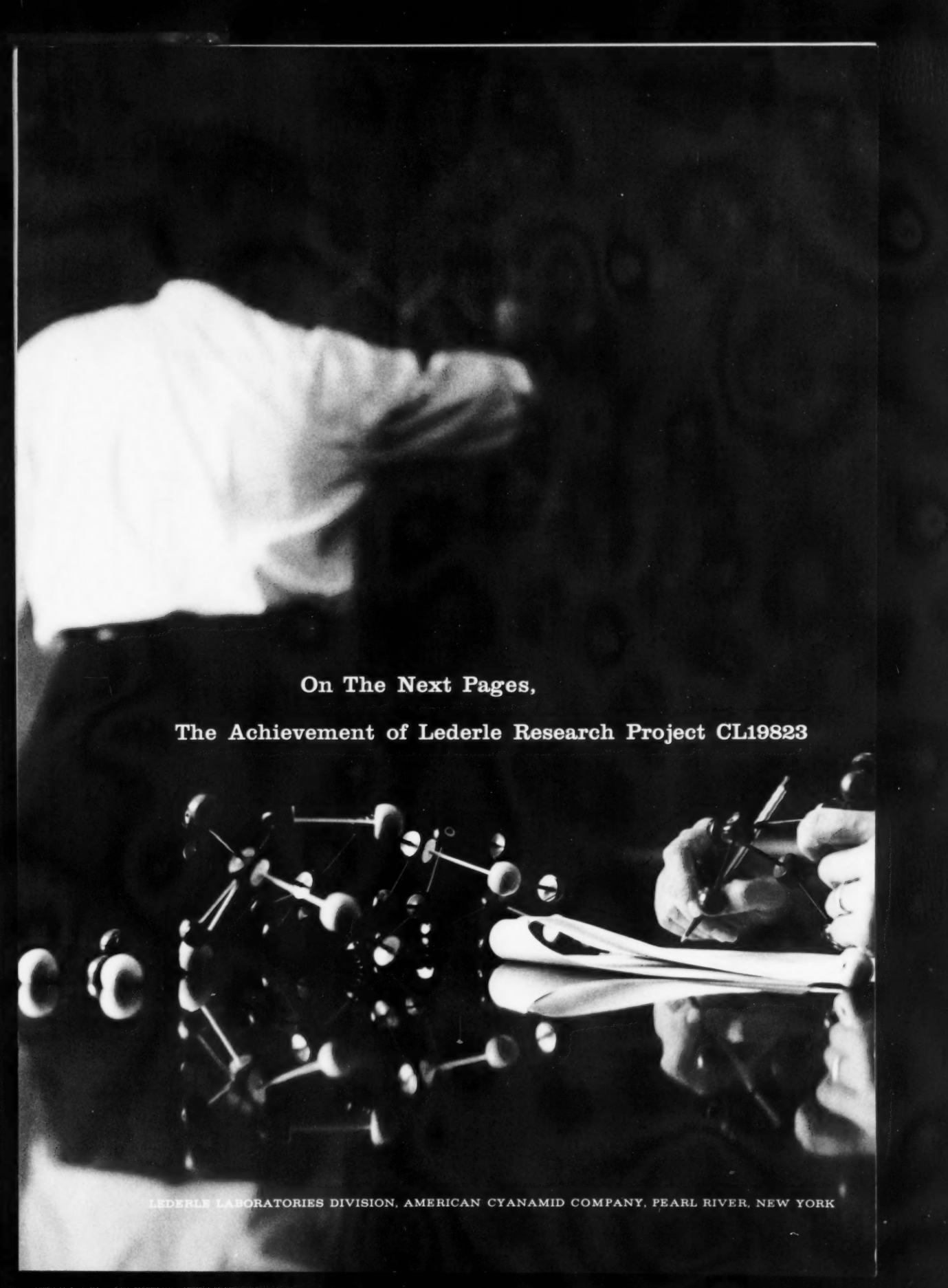
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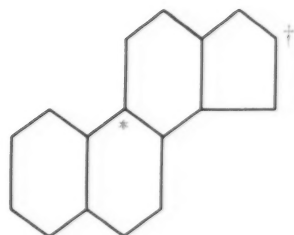
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- ◇ No interference with psychic equilibrium
- ◇ Low incidence of peptic ulcer and osteoporosis

# Biological Effects of Aristocort

with  
particular emphasis  
on:

## Kidney function

Animal studies on ARISTOCORT<sup>1</sup> have not demonstrated any interference with creatinine or urea clearance. Autopsy surveys of organs of animals on prolonged study of this medication have shown no renal damage.

## Sodium and water

ARISTOCORT produced an increase of 230 per cent of water diuresis and 145 per cent sodium excretion when compared to control animals.<sup>1</sup> Metabolic balance studies in man revealed an average negative sodium balance of 0.8 Gm. per day throughout a 12-day period on a dosage of 30 mg. per day.<sup>2</sup> Additional balance studies showed actual sodium loss when ARISTOCORT was given in doses of 12 mg. daily.<sup>3</sup> Other investigators observed significant losses of sodium and water during balance studies and that those patients with edema from some older corticosteroids lost it when transferred to ARISTOCORT.<sup>4,5</sup> In two studies of various rheumatic disorders (194 cases) on prolonged treatment, sodium and water retention was not observed in a single case.<sup>6,7</sup>

## Potassium and chlorides

There was no active excretion of potassium or chloride ions in animals given maintenance doses of ARISTOCORT 25 times that found to be clinically effective.<sup>1</sup> Potassium balance studies in humans<sup>2,3</sup> revealed that negative balance did not occur even with doses somewhat higher than those employed for prolonged therapy in rheumatoid arthritis. Hypokalemia, hyperkalemia or hypochloremia did not occur, when tested, in 194 patients with rheumatoid arthritis treated for up to ten and one-half months.<sup>6,7</sup>

## Calcium and phosphorus

Phosphate excretion in animals<sup>1</sup> was not changed from normal even with amounts 25 times greater (by body weight) than those known to be clinically effective. Human metabolic balance studies<sup>3</sup> demonstrated that no change in calcium excretion occurred on dosages usually employed clinically when the compound is administered for its anti-inflammatory effect. Even at a dosage level twice this, slight negative balance appeared only during a short period.

## Protein and nitrogen balance

Positive nitrogen balance was maintained during a human metabolic study on maintenance dosage of 12 mg. per day.<sup>3</sup> At dosages two to three times normal levels, positive balance was maintained except for occasional short periods in metabolic studies of several weeks' duration.<sup>2,3</sup>

There was always a tendency for normalization of the A/G ratio and elevation of blood albumin when ARISTOCORT was used in treating the nephrotic syndrome.<sup>8</sup>



### Liver glycogen deposition and inflammatory processes

An intimate correlation exists between the ability of a corticosteroid to cause deposition of glycogen in the liver and its capacity to ameliorate inflammatory processes.

In animal liver glycogen studies, relative potencies of ARISTOCORT over cortisone of up to 40 to 1 have been observed. Compared to ARISTOCORT, five to 12 times the amount of prednisone is required to produce varying but equal amounts of glycogen deposition in the liver.<sup>1</sup>

Most patients show normal fasting blood sugars on ARISTOCORT. Diabetic patients on ARISTOCORT may require increased insulin dosage, and occasional latent diabetics may develop the overt disease.

Anti-inflammatory potency of ARISTOCORT was determined by both the asbestos pellet<sup>1</sup> and cottonball<sup>9</sup> tests. It was found to be nine to 10 times more effective than hydrocortisone in this respect.

### Gastric acidity and pepsin

The precise mode of ulcerogenesis during treatment with corticosteroids is not known. There is much experimental evidence for believing this may be related to the tendency of these agents to increase gastric pepsin and acidity—and this cannot be abolished by vagotomy, anticholinergic drugs or gastric antral resection.<sup>10</sup> Clinical studies<sup>11</sup> of patients on ARISTOCORT revealed that uropepsin excretion is not elevated. Further, their basal acidity and gastric response to histamine stimulation were within normal limits.

### Central nervous system

The tendency of corticosteroids to produce euphoria, nervousness, mental instability, occasional convulsions and psychosis is well known.<sup>12</sup> The mechanism underlying these disturbances is not well understood.

ARISTOCORT, on the contrary, does not produce a false sense of well being, insomnia or tension except in rare instances. In the treatment of 824 patients, for up to one year, not a single case of psychosis has been produced. In general, it appears to maintain psychic equilibrium without producing cerebral stimulation or depression.

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# The Promise of Aristocort

## in Reduction of Side Effects

It is axiomatic to affirm that the undesirable collateral hormone effects of corticosteroids increase in frequency and severity the higher the dosage and the longer used.

It has also become well recognized that the most serious of the major side effects from long-term corticosteroid treatment are peptic ulcers, osteoporosis with fracture, drug psychosis and euphoria, and sodium and water retention leading often to general tissue edema and hypertension.

It is significant that of the close to 400 patients on the lower dosage schedules found effective in bronchial asthma and dermatologic conditions, only 1 case of peptic ulceration has developed. No other of the above side effects have been observed even though ARISTOCORT was administered continuously to them for periods as long as one year.

The treatment of rheumatoid arthritis with steroids appears to result in the highest incidence of side effects. For this reason, the side effects associated with ARISTOCORT therapy in 292 patients with rheumatoid arthritis are reported below.

### Peptic Ulcer

The occurrence of peptic ulcer in 292 patients with rheumatoid arthritis treated continuously for up to one year with ARISTOCORT is approximately 1 per cent (2 of the 3 occurred in patients transferred from prednisone). In the remaining 532 cases recently analyzed, only one ulcer has been discovered in a patient who apparently had no ulcer when he was changed from another steroid.

### Osteoporosis and Compression Fractures

The occurrence of osteoporosis with compression fracture in 292 patients with rheumatoid arthritis treated continuously for up to one year with ARISTOCORT is 0.33 per cent (1 case<sup>1</sup>). Although these results are encouraging, determination of the true incidence of osteoporosis will have to await the passage of more time.

### Euphoria and Psychosis

The euphoria so commonly produced by all previous corticosteroids has seemed a most desirable attribute to patients. In penalty, however, they have often later to pay for this by mental disturbances, varying from mild and transitory to severe depression and psychosis,<sup>2</sup> and toxic syndromes producing even convulsions and death.<sup>3</sup>

Since the onset of these complications is not directly related to duration of steroid administration,<sup>4</sup> the fact that not one case of psychosis occurred in 824 patients treated with ARISTOCORT, is most encouraging.

## Sodium Retention—Hypertension—Potassium Depletion

When 17 patients were changed from prednisone to ARISTOCORT, 11 rapidly lost weight although only one had had visible edema.<sup>5</sup> Sodium and water retention, hypokalemia or hyperkalemia and steroid hypertension did not appear in 194 rheumatoid arthritis patients treated with ARISTOCORT.<sup>1,6</sup>

The interrelation between blood and body sodium, and steroid hypertension has long been generally appreciated.<sup>7,8</sup> Except in rare instances, or when unusually high doses are used (e.g., leukemia), the problem of edema and hypertension caused by sodium and water retention, has been eliminated with ARISTOCORT.

### Minor Side Effects

Collateral hormonal effects of less serious consequence occurred with approximately the same frequency as with the older corticosteroids.<sup>1</sup> These include erythema, easy bruising, acne, hypertrichosis, hot flashes and vertigo. Several investigators have reported symptoms not previously described as occurring with corticosteroid therapy, e.g., headaches, light-headedness, tiredness, sleepiness and occasional weakness.

Moon facies and buffalo humping have been seen in some patients on ARISTOCORT. However, ARISTOCORT therapy, in many instances, resulted in diminution of "Cushingoid" signs induced by prior therapy. Where this occurs, it may be related to reduced dosage on which patients can be maintained.

### Reduction of dosage by one-third to one-half

In a double-blind study of comparative dosage in patients with rheumatoid arthritis,<sup>9</sup> 70 per cent of the cases were as well controlled on a dose of ARISTOCORT one-half that of prednisone. A general recommendation can be made that ARISTOCORT be used in doses two-thirds that of prednisone or prednisolone in the treatment of rheumatoid arthritis. There are individual variations, however, and each patient should be carefully titrated to produce the desired amount of disease suppression.

Comparative studies, of patients changed from prednisone, indicate reduced dosage of ARISTOCORT in bronchial asthma and allergic rhinitis (33 per cent),<sup>5</sup> and in inflammatory and allergic skin diseases (33-50 per cent).<sup>10,11</sup>

## General Precautions and Contraindications

Administration of ARISTOCORT has resulted in lower incidence of major serious side effects, and in fewer of the troublesome minor side effects known to occur with all previously available corticosteroids. However, since it is a highly potent glucocorticoid, with profound metabolic effects, all traditional contraindications to corticosteroid therapy should be observed.

No precautions are necessary in regard to dietary restriction of sodium or supplementation with potassium.

Since ARISTOCORT has less of the traditional side effects, the appearance of sodium and water retention, potassium depletion, or steroid hypertension cannot be used as signs of overdosage. As a rule patients will lose some weight during the first few days of treatment as a result of urinary output, but then the weight levels off.

Patients do not develop the abnormally voracious appetite common to previous corticosteroid administration. In fact, some patients experienced anorexia, and it is advisable to inform patients of this and to recommend they maintain a normal intake of food, with emphasis on liberal protein intake.

While precipitation of diabetes, peptic ulcer, osteoporosis, and psychosis can be expected to appear rarely from ARISTOCORT, they must be searched for periodically in patients on long-term steroid therapy.

Traditional precautions should be observed in gradually discontinuing therapy, in meeting the increased stress of operation, injury and shock, and in the development of intercurrent infection.

There is one overriding principle to be observed in the treatment of any disease with ARISTOCORT. *The amount of the drug used should be carefully titrated to find the smallest possible dose which will suppress symptoms.*

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# The Promise of Aristocort

## in Rheumatoid Arthritis

ARISTOCORT therapy has been intensely and extensively studied for periods up to one year on 292 patients with rheumatoid arthritis.

Significant is the fact that most patients were severe arthritics, transferred to ARISTOCORT from other corticosteroids because satisfactory remission had not been attained, or because the seriousness of collateral hormonal effects had made discontinuance desirable.

### Results of treatment

Freyberg and associates<sup>1</sup> treated 89 patients with rheumatoid arthritis (A. R. A. Class II or III and Stage II or III). Of these, 51 were on ARISTOCORT therapy from three to over 10 months. In all but a few patients, satisfactory suppression of rheumatoid activity was obtained with 10 mg. per day. Thirteen were controlled on 6 mg. or less a day, and for periods to 180 days. The investigators reported therapeutic effect in most cases to be A. R. A. Grade II (impressive) and that marked reduction in sedimentation rates occurred.

Another interesting observation in this study: Of the 89 patients treated, 12 had active ulcers, developed from prior steroid therapy. In six patients, the ulcers healed while on doses of ARISTOCORT sufficient to control arthritic symptoms.

Hartung<sup>2</sup> treated 67 cases of rheumatoid arthritis for up to 10 months. He found the optimum maintenance dose to be 11 mg. per day. Nineteen of these patients were treated for six to 10 months with an "excellent" therapeutic response.

### Dosage and course of therapy

The initial dosage range recommended is 14 to 20 mg. per day—depending on the severity and acuteness of signs and symptoms. Dosage is divided into four parts and given with meals and at bedtime. Anti-rheumatic effect may be evident as early as eight hours, and full response often obtained within 24 hours. This dosage schedule should be continued for two or three days, or until all acute manifestations of the disease have subsided, whichever is later.

The maintenance level is arrived at by reduction of the total daily dosage in decrements of 2 mg. every three days. The range of maintenance therapy has been found to be from 2 mg. to 15 mg. per day—with only a very occasional patient requiring as much as 20 mg. per day. Patients requiring more than this should not be long continued on steroid therapy.

The aim of corticosteroid therapy in rheumatoid arthritis is to suppress the disease only to the stage which will enable the patient to carry out the required activities of normal living or to obtain reasonable comfort. The maintenance dose of ARISTOCORT to achieve this end is arrived at while making full use of all other established methods of controlling the disease.

ARISTOCORT is available in 2 mg. scored tablets (pink); 4 mg. scored tablets (white). Bottles of 30.

### Bibliography

1. Freyberg, R. H., Bernsten, C. A., and Hellman, L.: Paper presented at International Congress on Rheumatic Diseases, Toronto, June 25, 1957.
2. Hartung, E. F.: Paper presented at Florida Academy of General Practice, St. Petersburg, Florida, Nov. 2, 1957.



# The Promise of Aristocort

## in Respiratory Allergies

○ About 200 patients with respiratory allergies have been treated with ARISTOCORT for continuous periods up to eight months.

### Results of treatment

Sherwood and Cooke<sup>1,2</sup> gave ARISTOCORT to 42 patients with bronchial asthma and allergic rhinitis. Average dose needed to control the asthmatic group was approximately 6 mg. per day (range, 2 to 14 mg.). Results, which were called "good to excellent" in all but four, were achieved on one-third less than similarly effective doses of prednisone or prednisolone.

The investigators noted other major improvements in ARISTOCORT therapy over the older steroids. There was no increase in blood pressure in any patient: *on the contrary, in 12 patients, there was reduction of pressure when they were transferred to ARISTOCORT.* One patient had required auxiliary antihypertensive drug therapy; over a nine-week period on ARISTOCORT, the pressure gradually fell from 206/100 to 136/79. In another case, the pressure slowly dropped from 205/105 to 154/86.

The number of cases in which these investigators tried ARISTOCORT in allergic rhinitis was not large enough to provide significant averages. However, the range of effective therapy was from 2 to 6 mg. per day. These strikingly low daily doses resulted in control of all signs and symptoms.

Schwartz<sup>3</sup> treated 30 patients with chronic, intractable bronchial asthma. At an average daily dose of 7 mg., he reported "good to excellent" results in all but one. Spies,<sup>4</sup> Barach<sup>5</sup> and Segal,<sup>6</sup> reported similar results at average daily maintenance doses of 4 to 10 mg. of ARISTOCORT.

### Dosage and course of therapy

The initial dosage range recommended is 8 to 14 mg. of ARISTOCORT daily. Although a rare, very severe case may require more than this on the first day of therapy, these dosages will usually result in prompt alleviation of dyspnea, wheezing and cyanosis. Patients are soon able to carry out a normal span of daily activity.

The maintenance level is arrived at by reduction of the total daily dose every three days in decrements of 2 mg.; in the over-all series, the average daily dose for bronchial asthma is approximately 8 to 10 mg. and for allergic rhinitis, 2 to 6 mg. per day. All total daily doses should be divided into four parts and given with meals and at bedtime. As in every condition where corticosteroids are employed, each patient's treatment should be individualized and the maintenance arrived at by careful titration against signs and symptoms of disease.

Patients with chronic bronchial asthma may require steroid therapy for several months. And since asthma may be associated with cardiac disease, especially in the older age groups, ARISTOCORT is particularly useful because of its ability to cause excretion of sodium and water.

ARISTOCORT is available in 2 mg. scored tablets (pink); 4 mg. scored tablets (white). Bottles of 30.

### Bibliography

1. Sherwood, H., and Cooke, R. A.: J. Allergy 28:97, 1957.
2. Sherwood, H., and Cooke, R. A.: Personal Communication.
3. Schwartz, E.: Personal Communication.
4. Spies, T. D.: Personal Communication.
5. Barach, A. L.: Personal Communication.
6. Segal, M. S.: Personal Communication.

 Lederle

# The Promise of Aristocort

## in Nephrotic Syndrome

◊ Fourteen patients with the nephrotic syndrome have been treated with ARISTOCORT for continuous periods of up to six weeks.

### Results of treatment

Hellman and associates<sup>1,2</sup> noted that ARISTOCORT, because of its favorable electrolyte effects, may well be the most desirable steroid to date in treatment of the nephrotic syndrome. However, thus far its use has been reported in only 14 children, of whom 8 had a complete diuresis and disappearance of all abnormal chemical findings. Four of the patients had diuresis, but continued to show some abnormal chemical findings, while two patients with signs of chronic renal disease failed to respond.

### Dosage and course of therapy

In order to produce maximal response, 20 mg. should be given daily until diuresis occurs. The dose should then be decreased gradually and maintained around 10 mg. a day. After the patient has been in remission for some time, it may be advisable to diminish the dose gradually and discontinue ARISTOCORT.

## in Pulmonary Emphysema and Fibrosis

◊ Eleven patients with pulmonary emphysema and/or fibrosis were treated with ARISTOCORT for continuous periods of over two months.

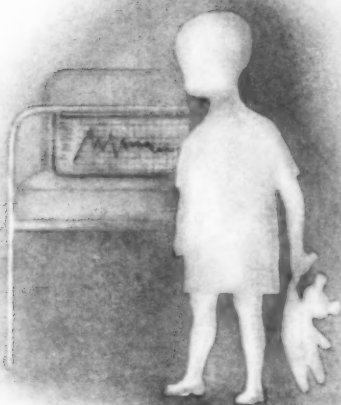
### Results of treatment

Only small series of cases observed by Barach,<sup>3</sup> Segal,<sup>4</sup> and Cooke,<sup>5</sup> are available. Barach treated patients who were not adequately controlled by prednisone, with the same dose of ARISTOCORT with significant improvement.

### Dosage and course of therapy

The initial suppressive dose range recommended is 10-14 mg. daily. Frequently, there is a prompt decrease in cyanosis and dyspnea, with increase in vital capacity.

The average maintenance dose level was 8 mg. a day. If it is desired to maintain a patient on continuous therapy for some months, dosages as low as 2 mg. a day have been successful. All decreases in dosage should be gradual and at a rate of 2 mg. decrements in total daily amount, every two to four days. The daily dosage is divided into four parts and given with meals and at bedtime.



## in Neoplastic Diseases

Forty-four children and adults have been given ARISTOCORT for palliative treatment of acute leukemia, chronic lymphatic leukemia, lymphosarcoma, lympholeukosarcoma and Hodgkin's disease.

### Results of treatment

Farber<sup>6</sup> has treated 22 children with acute leukemia for an average of three weeks. Of the 17 observed long enough to judge the efficacy of the medication, he rated five as excellent, three as good, two as fair and seven as poor responses.

Hellman and associates<sup>7</sup> gave ARISTOCORT to a group of patients with the various lymphomas in doses of 40 to 50 mg. a day—occasionally up to 100 milligrams. Treatment was continued in some cases for 17 weeks. Response was classified as good for the palliative purposes for which the drug was given.

### Dosage and course of therapy

Massive initial suppressive doses of 40 to 50 mg. per day in children (1 mg./kg./day) and up to 100 mg. a day in adults have been administered.

Responses to any specific dosage in these conditions vary so widely that only a general dosage range can be indicated. Treatment

must be individualized; rate of reduction in dosage and determination of maintenance levels cannot be categorized.

### Miscellaneous

Patients with various other diseases have been treated by several clinical investigators. These include patients with osteoarthritis, acute bursitis, rheumatic fever, spondylitis, other "collagen-vascular" diseases (dermatomyositis, etc.), thrombocytopenic purpura, chronic eosinophilia, hemolytic anemia, diuretic-resistant congestive heart failures, and adrenogenital syndrome.

There have not been sufficient patients in any of the above categories to permit definitive treatment schedules to be finally established for ARISTOCORT. Additional studies are now in progress and physicians desiring information on any of these diseases are requested to write to Lederle Laboratories, Pearl River, New York for available data.

ARISTOCORT is available in 2 mg. scored tablets (pink); 4 mg. scored tablets (white). Bottles of 30.

### Bibliography

1. Hellman, L., Zumoff, B., Kretshmer, N., and Kramer, B.: Presented at Nephrosis Conf., Bethesda, Md., Oct. 26, 1957.
2. Ibid: Personal Communication.
3. Barach, A. L.: Personal Communication.
4. Segal, M. S.: Personal Communication.
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6. Farber, S.: Personal Communication.
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# The Promise of Aristocort

## in Inflammatory and Allergic Skin Diseases

Over 200 patients with allergic and inflammatory skin diseases (including psoriasis, atopic dermatitis, exfoliative dermatitis, pemphigus, dermatitis herpetiformis, eczematoid dermatitis, contact dermatitis and angioneurotic edema) have been treated continuously with ARISTOCORT for periods of up to eight months.

### Results of treatment

Rein and associates<sup>1</sup> treated 26 patients with severe dermatitis. Twenty-four had been on prednisone when changed to ARISTOCORT. While some had found satisfactory symptomatic relief, others had also developed side effects—moon face, buffalo hump, increased appetite with excessive weight increases and gastro-intestinal disturbances.

These investigators determined the equivalent dosage of ARISTOCORT to be approximately two-thirds that required to control symptoms on the previous corticosteroid. Thirteen of the 26, who had developed moon face, noted either an actual decrease or no further increase when transferred to ARISTOCORT. In addition: *Voracious appetites disappeared, with loss of weight in 11 patients; there was no elevation in blood pressure, and no necessity to restrict sodium or administer supplemental potassium.* Sherwood and Cooke,<sup>2</sup> and Shelley and Pillsbury<sup>3</sup> obtained similar results in allied disorders.

Hollander<sup>4</sup> first observed that ARISTOCORT appears to have striking affinity for the skin and great activity in controlling such diseases as psoriasis, for which other corticosteroids have been indifferently effective. Shelley and Pillsbury,<sup>3</sup> in 50 cases of acute extending psoriasis found that over 60 per cent were markedly improved.

### Dosage and course of therapy

The recommended initial suppressive dose range is 14 to 20 mg. per day. In very severe cases, temporary dosages up to 32 mg. a day

have been successfully employed. Once lesions are suppressed, gradually reduce dose to the maintenance level—which may be as low as 2 mg. per day.

### Bibliography

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## in Disseminated Lupus Erythematosus

Forty patients with disseminated lupus erythematosus were treated with ARISTOCORT for continuous periods of up to nine months.

### Results of treatment

Patients have responded very promisingly to therapy. Dubois<sup>1</sup> has had the largest single experience (28 cases) with ARISTOCORT in the treatment of this disease. He reported 25 of the 28 responded favorably.

Freyberg,<sup>2</sup> Hartung,<sup>3</sup> Hollander,<sup>4</sup> Spies,<sup>5</sup> and Segal,<sup>6</sup> each in smaller series of cases, reported similarly good therapeutic responses.

### Dosage and course of therapy

The initial suppressive dose recommended is 20-30 mg. daily. Once the desired effect is achieved, the dose should be reduced gradually to maintenance levels (3 to 18 mg. per day).

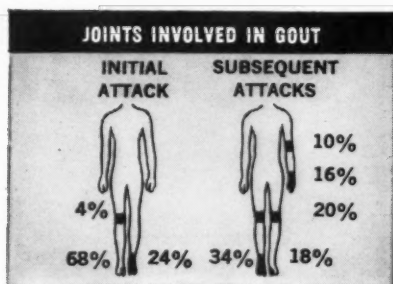
In severely ill patients large doses may be required for several days in order to preserve life. Even on these large doses, edema and sodium retention have not occurred.

ARISTOCORT is available in 2 mg. scored tablets (pink); 4 mg. scored tablets (white). Bottles of 30.

### Bibliography

1. Dubois, E. L.: Personal Communication. 2. Freyberg, R. H.: Personal Communication. 3. Hartung, E. F.: Personal Communication. 4. Hollander, J. L.: Personal Communication. 5. Spies, T. D.: Personal Communication. 6. Segal, M. S.: Personal Communication.

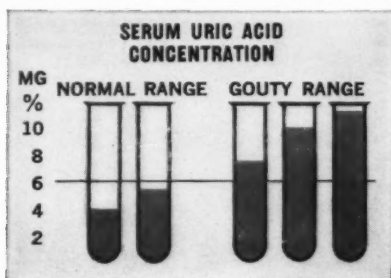




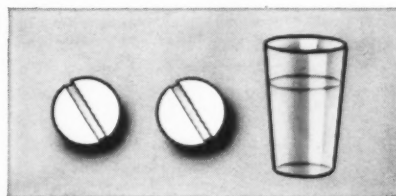
1. Recurrent joint pain followed by long periods of complete remission. (Percentages refer to incidence.)



2. Enlargement of bursae such as in this case involving the olecranon bursa.



3. Elevated serum uric acid levels.



4. Colchicine test: full dose (0.5 mg.) every 1 to 2 hours until pain is relieved or nausea, vomiting or diarrhea occur. The test requires usually 8 to 16 doses. Pain relief is highly indicative of gout.

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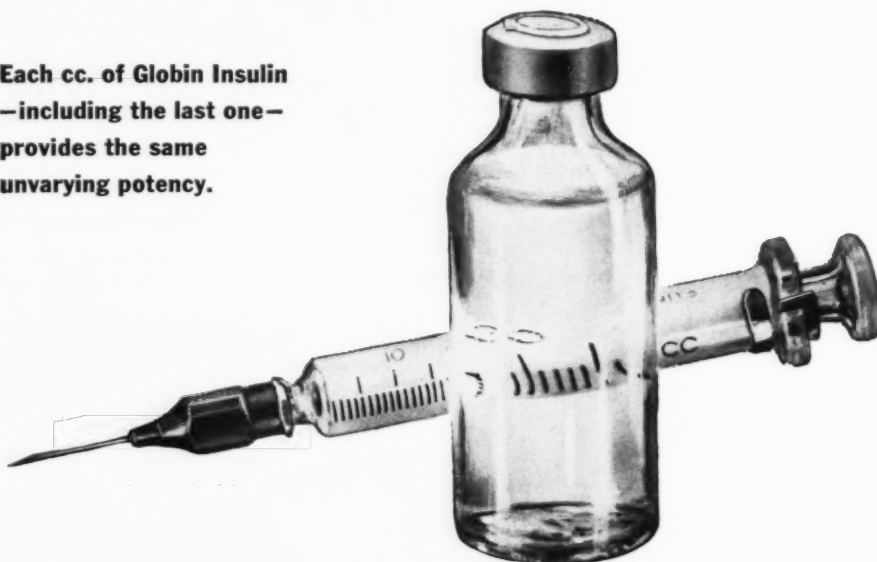


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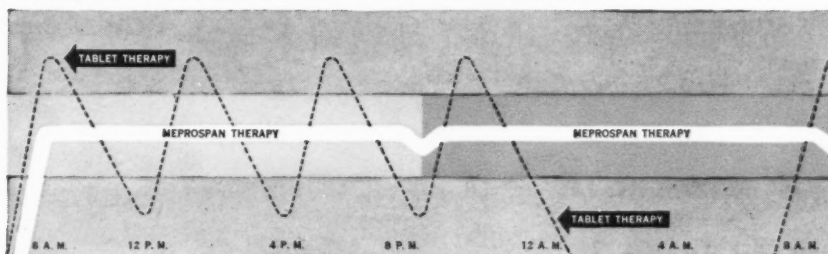
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1. Russek, H. I.: Postgrad. Med. 19:562 (June) 1956.

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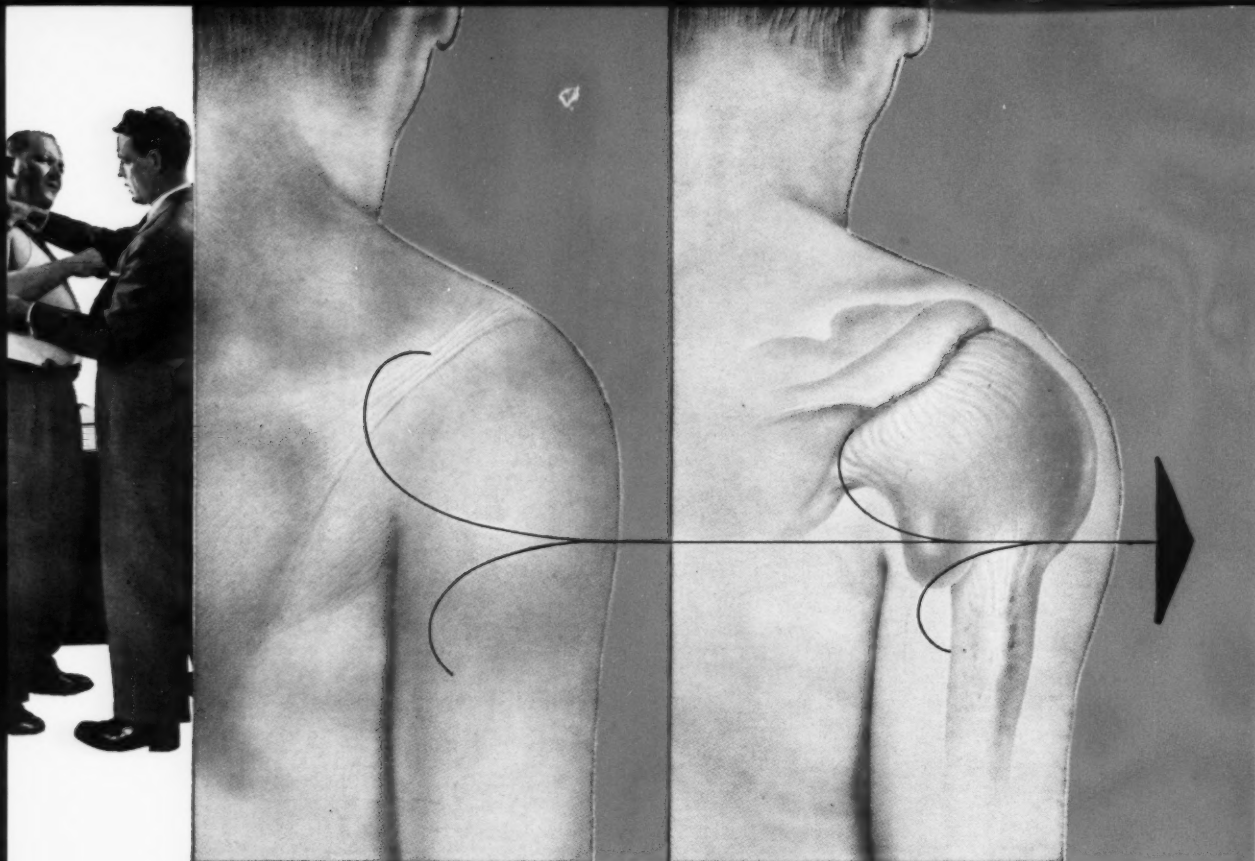
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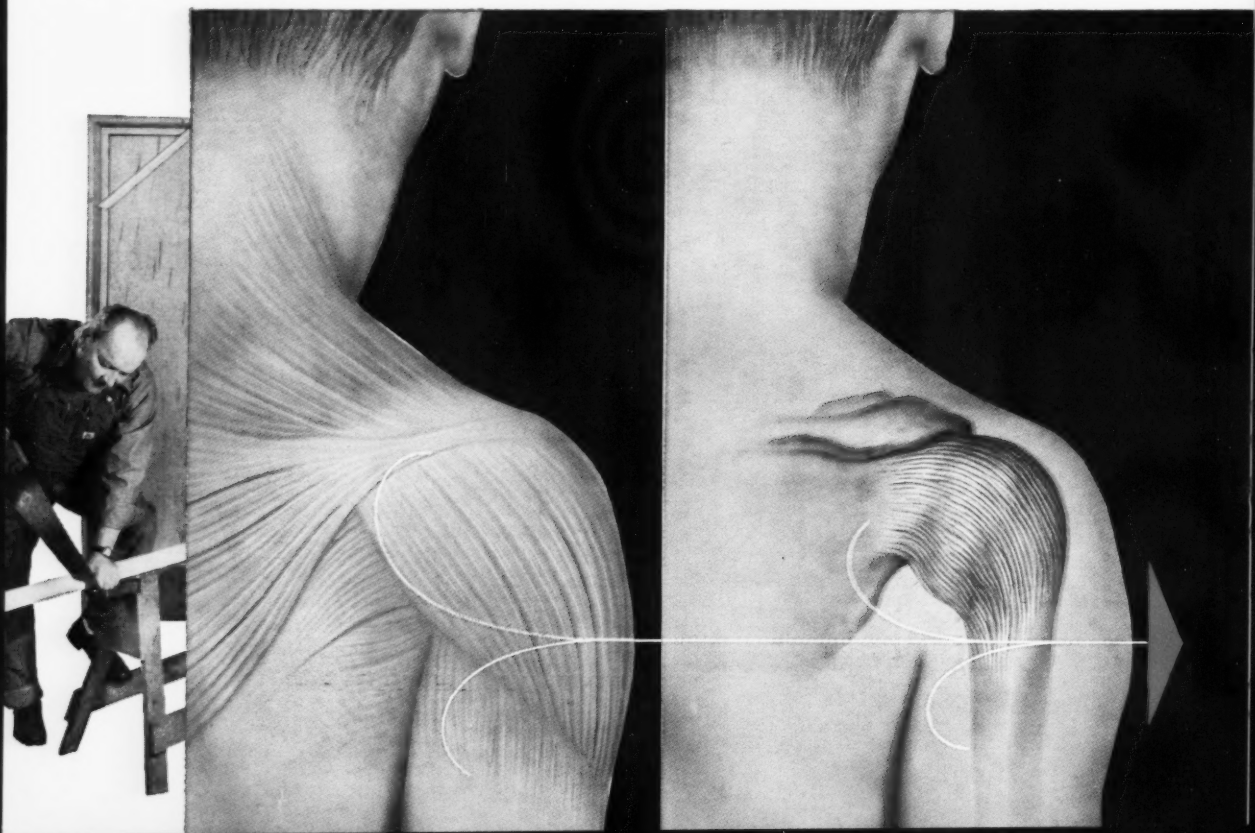
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1. Comroe's Arthritis: Hollander, J. L., p. 149 (Fifth Edition, Lea & Febiger, Philadelphia, Pa. 1953).  
2. Merck Manual: Lyght, C. E., p. 1102 (Ninth Edition, Merck & Co., Inc., Rahway, N. J. 1956).

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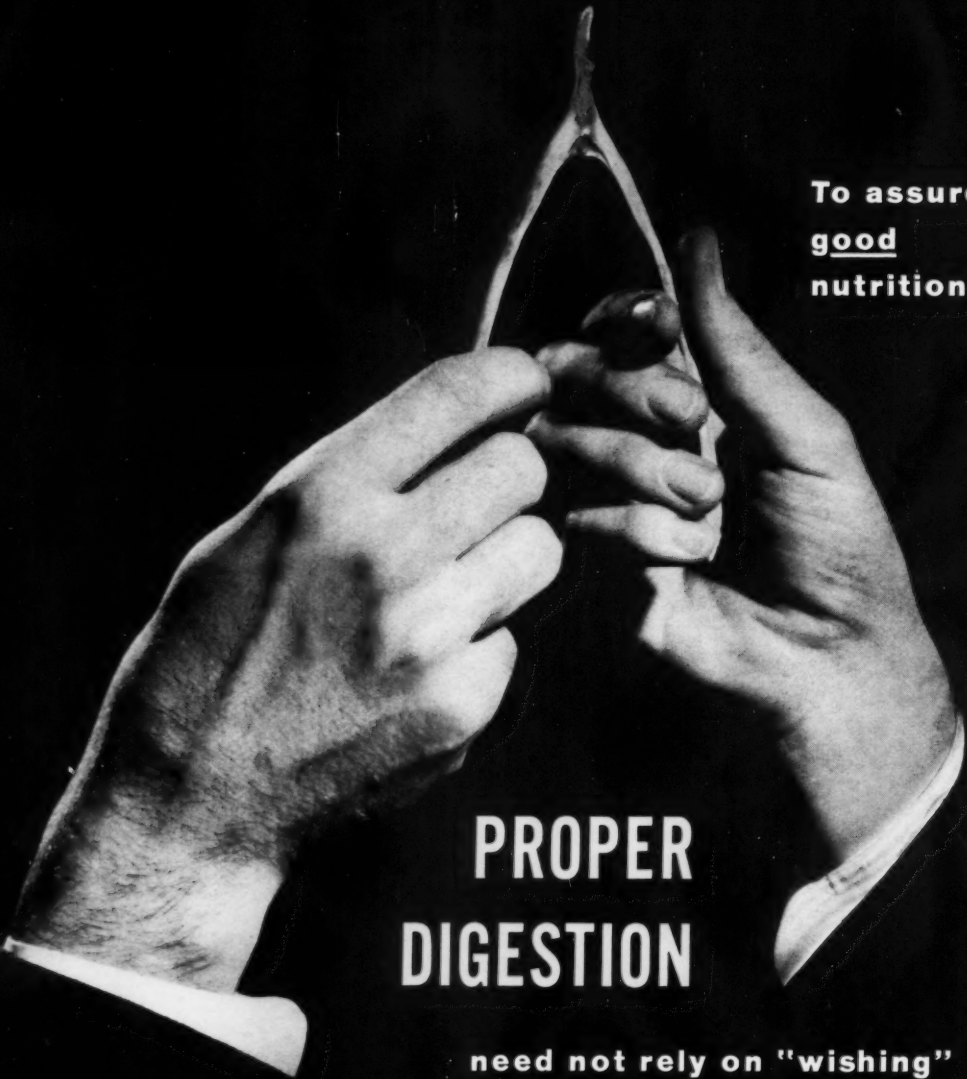
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base or the hydrochloride alone. In addition, the average levels derived from the tetracycline base of the chlortetracycline base were higher than those produced by the corresponding hydrochloride though lower than those resulting from the mixture containing the base and sodium metaphosphate. In the study with chlortetracycline<sup>6</sup> capsules containing a mixture of the hydrochloride and sodium metaphosphate were also included in the crossover, and the average levels produced by these capsules were the same as with the mixture of chlortetracycline base with sodium metaphosphate.

Although the enhancement of blood levels of tetracycline by phosphate, either complexed to the tetracycline or mixed with the base or the hydrochloride, thus seemed fairly well established, some doubts still remained because certain reliable observers (including many whose results have not been published) failed to confirm the findings with the materials and methods they used. Further confusion seemed to be added by a subsequent report of Welch et al.,<sup>7</sup> who, in repeating a crossover study with capsules of tetracycline phosphate complex and tetracycline hydrochloride with and without sodium phosphate, found

antibacterial activity than was observed in their absence. Oil and sorbitol did not interfere with tetracycline absorption.

Dicalcium phosphate is widely used as a filler in various capsules, including those of the tetracyclines. The authors cite a large number of other studies that implicate the presence of calcium ions as the cause of the reduced absorption of tetracyclines and show that citric acid can partially neutralize this effect. The depressing effect of food on the serum levels of tetracycline is likewise explained by the goodly amount of minerals contained in commercial laboratory diets, and they postulate that the multivalent cations may be responsible for the poorer absorption of the drug. The authors could not explain the failure of citric acid to enhance serum concentrations when administered with tetracycline base in contrast to its marked effect when given as the hydrochloride. However, they hypothesized that the ability of citric acid to enhance serum levels of tetracycline is related to its ability to form complex salts with the tetracycline.

...Tetracycline hydrochloride and citric acid, in an encapsulated mixture, produced higher serum concentrations and greater urinary excretions, and hence better absorption of tetracyclines, than any other preparation studied...

...endum to the last mentioned paper of ... et al.<sup>7</sup> indicates that in their study the capsules of tetracycline hydrochloride, chlortetracycline hydrochloride and tetracycline phosphate complex all contained dicalcium phosphate as a filler, whereas the capsules containing citric acid and sodium hexameta-phosphate did not contain any dicalcium phosphate. This could clearly explain the discrepancies noted in that study.<sup>7</sup> Likewise, the inconsistencies in other studies may very well have been due to the use of

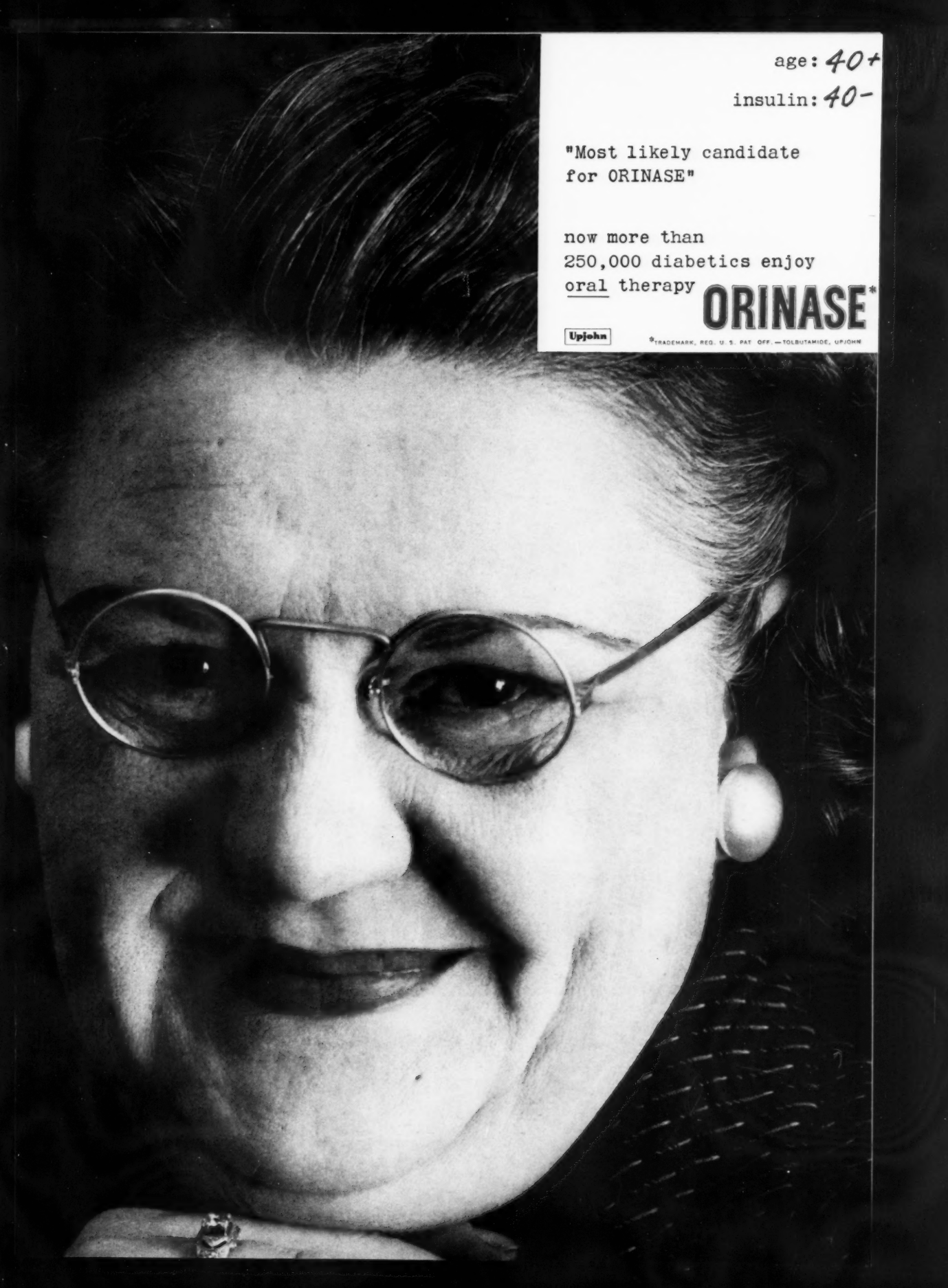
Editorial.  
*The New England Journal of Medicine.*  
258:97-99, (January 9) 1958.

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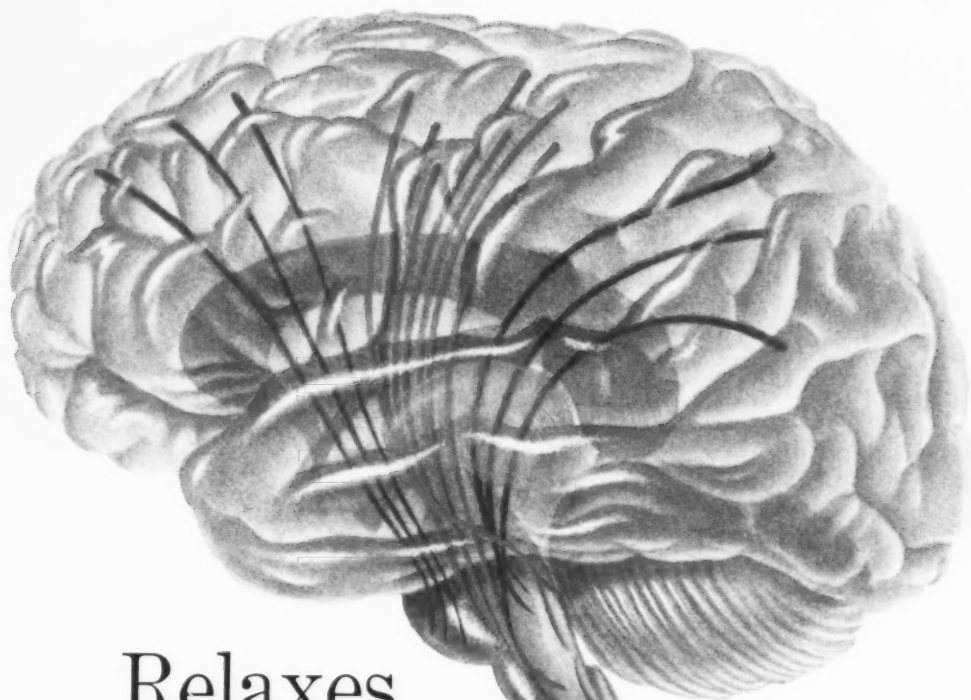
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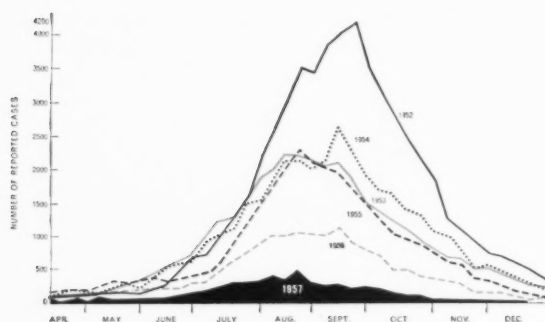
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1. J. A. M. A., 165:21 (November 23), 1957.

2. Department of Health, Education, and Welfare: News Release, October 10, 1957.

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# DELAWARE STATE MEDICAL JOURNAL

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## OMENTAL INFARCTION

EDWARD M. BOHAN, M.D.\*

### CASE HISTORY:

The patient, a man, age 32, was received into St. Francis Hospital on April 20, 1953. He was complaining of severe pain in the right upper quadrant of the abdomen. His family doctor suggested that he might have acute cholecystitis or a ruptured ulcer. His pain was relieved for 24 hours previous to admission. The patient stated that it seemed like a pressure pain. Slight diarrhea was present. The patient's past medical history consisted of bronchial asthma, which was present since early childhood, and an attack of amoebic dysentery while in Germany during World War II.

The patient was 6' 3" tall; his weight was 205 pounds. His blood pressure was 164/82. There was no icterus. Examination was normal except for tenderness in the right upper quadrant just below the ribs. No edema or varicose veins of the extremities were found.

The white blood count was 16,650, with 82% polys. Blood sugar, urea nitrogen, and prothrombin time were normal. Icterus index was 3 units and van den Bergh reaction was negative for the direct and indirect phases. Urinalysis was negative for albumen and sugar, and showed 8 to 10 red blood cells per high powered field. No casts or white cells were seen.

A surgical consultant made a diagnosis of mild cholecystitis, rather than appendicitis, although the possibility of an appendix in the retrocecal location as the cause of the trouble was kept in mind. However, the patient's condition became gradually worse. His temperature was elevated to 99.4° oral-

ly, and a decision was made to operate on the next day.

Two more blood counts taken showed no change.

An x-ray of the chest showed normal heart and lungs and a film of the abdomen showed normal psoas and kidney shadows on both sides. There was no evidence of any radiopaque calculi in the gall bladder region or in the urinary tract. Normal distribution of gas shadows and soft tissue shadows was observed.

### SURGICAL FINDINGS:

The greater omentum in the right upper abdomen showed localized hemorrhagic necrosis about 3" x 4" in size, with a slight amount of peritoneal blood-tinged fluid covering this area. There were no plastic adhesions and there was no gross evidence of torsion of the omentum. The involved segment of the greater omentum was directly under the parietal peritoneum and right rectus muscle halfway between the costal margin and level of the umbilicus, and the abdominal wall showed no evidences of trauma and no signs of hemorrhage. The liver and gall bladder were normal, and the appendix was found in the retrocecal position in the right lower quadrant, distended with a fecalith about the size of a pea in its mid-portion. There was no other abnormality detected in the adjoining bowels and mesentery, and there was no evidence of internal hernia.

The pathological report by Dr. Joseph W. Abbiss was as follows: "Gross examination reveals two portions of what appear to be fatty tissue which are markedly engorged with blood. Histological examina-

\* Chief in Medicine and Metabolism, St. Francis Hospital, Wilmington, Delaware.

tion reveals fatty omental tissues which are markedly engorged with blood and which are showing early gangrenous changes. Diagnosis: gangrenous omentum."

The appendix was also removed at the time of operation, and showed fibrosis.

The patient was readmitted to St. Francis Hospital on December 7, 1957, because of abdominal pain and vomiting. Operative procedure at this time consisted of removal of a band of adhesions in the right pelvis which was causing obstruction of the ileum. Recovery was uneventful.

Tille<sup>1</sup> was able to collect records of only 26 cases of this type up to 1953. In August, 1957, Henshaw and Stafford<sup>2</sup> reported the case of a child. A case was reported by O'Beirn.<sup>3</sup> He noted in his case that the pain was aggravated by deep breathing and bending. In 1950, Manfredi<sup>4</sup> reported a case of omental segmental infarction in infancy. He stated that only one case in childhood had previously been reported. Mitchener<sup>5</sup> reported cases in December, 1954. James<sup>6</sup> reported two cases of acute segmental infarction due to primary torsion. He stated that 145 cases of infarction due to primary torsion had been reported up until 1951.

In this particular case, there was no torsion. The patient was a young, healthy man, only slightly obese.

The differential diagnosis requires the physician and surgeon to be conscious of this rare entity in their mental classification of the causal factors of acute abdomen.

Once the abdomen has been opened, the sero-sanguinous fluid will make the surgeon suspicious of omental infarction. James also emphasizes this point in his two case reports.

#### SUMMARY

A case of omental infarction with gangrene of the greater omentum has been reported. No torsion was present. Less than 35 cases of this type have been reported in the literature. The appendix was not in an active pathological state at the time surgery was performed; the gangrenous portion of greater omentum was removed.

The patient returned to St. Francis Hospital four years later with intestinal obstruction due to adhesions. Surgery was performed to relieve this condition. Recovery was uneventful.

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## CASE REPORTS OF THE SURGICAL DEPARTMENT OF ST. FRANCIS HOSPITAL

Edited by J. F. HUGHES, M.D.

### CASE 4317

**HISTORY:** This patient was admitted in October, 1957 and discharged 19 days later; she was 42 years of age. Her main complaint on admission was pain in the right lower quadrant and nausea. She has quite an interesting past history; a "stroke" in August, 1950, a mitral valvectomy in 1952 at a hospital in Philadelphia, and she has had three Cesarean sections, one in 1953, another in 1954 and the last one in 1956.

She stated that "on and off" she had pain localized in the right lower quadrant for quite some time. Since two days prior to admission, she had this pain and it became progressively worse. She was nauseated on several occasions.

**PHYSICAL EXAMINATION:** There was no cardiac enlargement, sinus rhythm was normal, rate was regular with sinus extrasystole. A grade 3 systolic harsh murmur radiated to the other areas, especially toward the left. The lungs were clear to palpation, percussion and auscultation. Liver, spleen and kidneys were not palpable. She was very tender to palpation on the right lower quadrant, with abdominal guarding and some rebound tenderness. There was a scar on the midline below the umbilicus. There was a partial impairment of movement of the left leg but there was no edema of either extremity, nor were there any varicose veins.

Impressions were (1) Appendicitis, acute; (2) Mitral insufficiency; and (3) Hemiplegia, left.

**DR. L. JONES:** This woman, as Dr. Graff will tell you, had a kind of bluish-grey color and looked very, very ill. I might add that Dr. Graff told me that he had been anes-

thetist for all three of these sections, and it was "touch and go" each time, whether she'd make the grade.

I made a right rectus incision, and I broke into an appendicial abscess. I removed the appendix and aspirated the abscess and placed a little sulfanilamide powder in the abscess cavity and placed a drain. She was removed from the operating table in rather poor condition.

**DR. P. OLIVERE:** On the fifth post-operative day this patient had a portable film of the chest which was taken in the supine position. The film showed extensive opacity and impaired aeration at the right base with depression of the short transverse fissure. Changes at the right base gave the appearance suggesting atelectasis, although I felt that an underlying pneumonia could not be ruled out.

There was a partially opaque rubber catheter passing down into the stomach. Outside of that, the lung fields looked within normal limits.

**DR. J. GRAFF:** This patient was not intubated at the time of surgery. At the time of her first Cesarean section, she exhibited signs of a stroke, heart failure and was a poor risk. At that time, we used an abdominal wall block, and it was a perfectly satisfactory procedure. She had good anesthesia and she cooperated well.

At the next Cesarean, we attempted the same thing, and this was just about as unsuccessful as the first was successful. We ended up by giving her general anesthesia which she tolerated fairly well. In the third Cesarean section, we decided to just go ahead and give her general anesthesia which we did right from the start, and again we

did well. It is a question of depth of anesthesia more than anything else. I don't know that choice of anesthesia particularly presents a problem here.

DR. JONES: As her post-operative course progressed her chest symptoms subsided and she was discharged in good condition.

#### CASE 4375

HISTORY: This 64 year old woman was admitted in November, 1957, and discharged 23 days later. Her chief complaint was bleeding from the rectum with resultant weakness. She was admitted because of the rectal bleeding. Color of the blood was dark and the patient felt very weak. She stated that these symptoms were present since the day prior to admission. She also stated that she had had some episodes of rectal bleeding a few years ago and was admitted here and several blood transfusions were given to her.

PHYSICAL EXAMINATION: There were no especially significant findings except for the bleeding.

The operative report reads as follows: "This patient has had repeated hemorrhages, associated with shock since admission, with the passage, each time, of dark liquid blood and blood clots by rectum. Upon opening the abdomen, the colon was found to be moderately distended by gas and liquid dark blood, semi-solid blood and blood clots, especially in the region of the descending colon and the upper portion of the sigmoid colon. The distal part of the colon appeared to be essentially empty, without evidence of much blood in this area. The colon on the right side of the abdomen did not appear to contain blood. It is, therefore, presumed that the bleeding site was probably in the region of the splenic flexure.

Careful external exploration of the colon failed to reveal any gross evidence of a lesion which would account for the bleeding. Similarly, internal exploration of the colon, using the sigmoidoscope, did not reveal any evidence of active bleeding, ulcer or other lesion to account for the repeated hemorrhages. No active bleeding or bright red blood was seen at the time of explora-

tion. The small intestine appeared to be mildly gas-distended, but not obstructed and showed no evidence of containing blood. The stomach and duodenum were empty. No evidence of bleeding from the upper intestinal tract was found. No evidence of peptic ulcer was found on examination, of the stomach and duodenum. The lesion from which the hemorrhages occurred into the colon is still not known. No evidence of ulceration or diverticulitis was found within the lumen of the bowel on internal examination of the colon. No evidence of malignant disease was seen. The patient's pulse and blood pressure remained within normal range throughout the operation."

DR. P. OLIVERE: The original film taken shortly after admission, showed some generalized distension of loops of colon, as well as some small intestinal loops. This generalized distension gave more the appearance of an ileus, rather than of an obstructive lesion. She was quite distended at this time. She had a metal tube drain (this was probably after her original surgery) in the pelvic portion of the abdomen.

On the 12th post-operative day portable films taken of the chest in a sitting position in bed showed that there was impaired aeration and clouding over both bases. These changes appeared more marked at the right base, with some relative elevation of the right diaphragm. We felt that this represented a hypostatic pneumonia, involving the bases.

Six days later there was almost clearing of the lung changes and better aeration of both lungs at this time, although there was some clouding obscuring the left costophrenic angle, suggesting a mild amount of effusion in the left base. We felt that there was clearing of the pneumonic changes at the bases with some residual effusion at the left base.

DR. C. SMITH: This patient presents a rather common problem, it seems, of bleeding from the intestinal tract without apparent cause. She had had previous hemorrhages and had studies of her upper and lower intestinal tract when she was not bleeding and no lesion was found.

As you all know, the principal causes of gastro-intestinal hemorrhage are rather easy to identify. When you do not find one of them, however, you are left with some rather bizarre possibilities. Just to mention one of them, which Dr. C. W. Mayo has called attention to, jejunal diverticulum, a lesion that bleeds quite commonly but one which might well not be found in the upper or lower gastro-intestinal tract x-rays. Small bowel x-rays, I feel, should be done on this patient even though they are a lot of work and not too reliable.

A patient like this one always brings you back to what Dr. Harvey Stone published some years ago. This paper is not out of date and was quoted from the Lahey Clinic as recently as two years ago. Dr. Stone had the courage to publish a series of patients who had gastro-intestinal hemorrhages on whom he was unable to find a cause for bleeding at the time of operation. He felt that it may not be worthwhile operating on such patients unless the magnitude of the bleeding demands it, and such appears to have been the circumstances here.

I don't say the foregoing in criticism of anything that has been done on this patient. I merely wish to point out the problem that we are discussing has been present for many years. I believe, however, that some change in policy has been made. Dr. L. K. Ferguson, a few years ago, published a few cases in which he was able to identify and treat the site of bleeding during the hemorrhage. He recommended that this procedure be carried out in selected cases. We all know that it is very difficult to find a bleeding point when the intestinal tract is filled with blood.

DR. D. PRESTON: When the patient left the hospital, I asked her if she would let me re-examine her in about four weeks. Realizing that she was still weak and had had much hospital treatment, I didn't wish to add to her inconvenience by extensive examination at the time. It is my thought that we may find an organic lesion to account for the hemorrhage which she had, but at present, I don't know the cause. It's very disturbing to have a patient in a dan-

gerous shock condition from hemorrhage and not be able to control the source of the bleeding. It is possible that she may have a superficial lesion in the mucosa that is not easily seen by direct inspection, perhaps a superficial erosion of a vein in the mucosal layer of the colon. I am hopeful, of course, that she will not have another hemorrhage, but we have no reason to feel secure in that thought because it is possible that the problem may be repeated for us.

DR. C. SMITH: In order that I may appear not to know too much about this problem, I wish to state that I have at present, under my care, a patient who has had four hemorrhages from his intestinal tract. This patient has had two upper gastro-intestinal tract x-rays, two barium enemas and a small bowel x-ray. I still don't have a diagnosis, and I am not going to do anything to him unless I am forced to it, which I may well be. Once again, I wish to point out that it is not uncommon to be unable to identify, prior to operation, the cause of a gastro-intestinal hemorrhage.

DR. ARMINIO: This problem also occurs in the younger age group. I have one now that I've explored twice, who, on both occasions, unfortunately was rather drastic, and we thought we couldn't control the bleeding. This child went on and expired, and at autopsy no reason for the bleeding was found either. So, this problem may continue and unfortunately, may go on to the very end, and you still have no diagnosis.

#### CASE 4019

SUMMARY: This patient had a subtotal gastrectomy for ulcer symptoms and seven days after the operation the patient had a massive hemorrhage.

DR. D. PRESTON: This patient is a 59 year old stocky, white man. He has had repeated upper gastro-intestinal hemorrhages during recent years. He is known to have had a chronic peptic ulcer for which he'd been treated constantly, except for the times when he would go off his diet and drink more whisky than he should.

The immediate problem at the time of the current admission was control of the hemorrhage, which was done by conservative treatment. After the blood volume had been restored by transfusion, the question then arose as to whether he should have a resection of the stomach because of the strong possibility of future hemorrhage, realizing that the risk each time was becoming greater. He had had five previous hemorrhages. There had been multiple admissions for transfusions to correct low red blood values following the vomiting of blood.

After removing about 75% of the stomach, we opened the specimen and found a small ulcer in the central portion of it on the posterior wall, which was assumed to be the cause of his hemorrhages. The pathologist examined this tissue and reported adenocarcinoma. It would seem that the resection was adequate, presuming this to be an early malignant change in a previous benign peptic ulcer of the stomach.

In addition to the ulcer problem, and carcinoma problem, there was also chronic hypertrophic gastritis, as evidenced by large rugose folds of mucosa, which upon examination by the pathologist, showed the typical round cell infiltration characteristic of this disease. The site of his hemorrhage one week after resection was not certain, although we presume that it was from the residual upper gastric segment, being about 25% at the gastric cardia. The cause of the bleeding is thought to be residual chronic hypertrophic gastritis which persists in this retained segment. It is possible that he could have bled from the suture line, although this would seem unlikely to occur seven days after operation. No duodenal ulcer was present at the time of resection, so it was not a problem here of leaving ulcer disease in the duodenum, which might be considered a possible source for bleeding. His liver appeared normal and no evidence of portal hypertension was seen at the time of operation.

Of course, we cannot exclude the possibility that he may have bled from other portions of the intestinal tract below the duodenal level. I'd be grateful for sugges-

tions as to further treatment, either medical or surgical. Should other investigative measures be undertaken to determine whether he has another organic lesion, perhaps, to cause this post-operative hemorrhage? He has been doing well and has had no further complication since his discharge from the hospital.

DR. P. OLIVERE: This man has had several studies of the stomach and they showed marked thickening of the rugal folds, especially in the antrum, but no evidence of any constant defect. Peristalsis could be seen to pass through all the way through, fluoroscopically, and we have films here to show it. The cap and duodenal loop looked all right. There were numerous diverticuli in the colon.

There is the appearance of the thickening of the rugal folds in the region of the antrum. There are no changes in the loop.

We got a good study which shows the esophagus well-outlined, and without much trouble, I think we could call this esophagus normal, with no evidence of any diverticuli or any varices.

This film shows the same thickening throughout the stomach, especially in the antrum, but peristaltic waves can be seen going through. The ulcer was never shown on the film. The only thing we did see was the marked thickening of the rugal folds, but peristalsis did go through, and the esophagus looked normal.

This study was taken after the operation, showing about 20 to 25% of the stomach contained in the cardiac end, with no changes suggesting recurrent jejunitis or evidence of the "dumping syndrome", and there was no evidence of any obstructive signs.

#### CASE 4028

HISTORY: This is another patient that had a subtotal gastrectomy for a simple peptic ulcer. He was 47 years of age and developed pneumonia and empyema associated with post operative abdominal abscess. (Figure 1.)

DR. D. PRESTON: His chief complication was not the duodenal fistula. That was

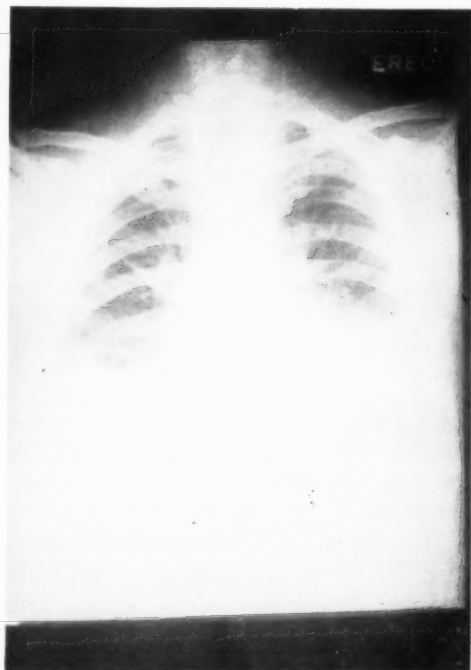


FIGURE 1

Changes in left lower pleural space suggest collection of fluid.

easily controlled by getting a tube in for drainage, but he nearly died of post-operative pneumonia and empyema. He apparently had accumulated a considerable quantity of gastric fluid during 12 hours of fasting before operation. He inhaled vomitus during induction of his anesthesia and that gave the anesthetist a rather rough time for a while. With a tube in the trachea he suctioned out as much of the acid-gastric fluid as he could, and the operation continued without event. After the operation the patient developed a high fever, rapid pulse rate and dyspnea with clinical evidence of a pneumonitis. He was given oxygen, symptomatic treatment, antibiotics and after a period of time, his temperature subsided to normal.

He went home, developed a high fever after several days, and came back. We put a tube in his left thorax and drained off four hundred cc. of pus. His temperature stayed down and he left the hospital. I saw him again later. He was having pain in his left chest. X-ray film showed

a shadow at the base of the lung on the left side, which the radiologist did not think was an abscess but believed it to be a zone of pneumonitis. He has now been admitted to another hospital because of bed shortage at St. Francis Hospital. His temperature remains normal, but it is thought that he may have some residual inflammatory changes in the base of his left lung. The abdominal incision has long since healed, and he is on a general diet, but I think Dr. Graff might wish to warn us about preparing our patients better for operation. In this particular instance, there was stenosis of the outlet of the stomach which may have accounted for an over-accumulation of fluid in the stomach, and I believe that this is a serious matter, because it could have resulted in this man's death. Inhaling of vomitus may have been preventable. If a tube had been put down and gastric juices removed by suction before sending him to the operating room, we might not have had the risk of this serious complication.

#### CASE 3930

**HISTORY:** This case is a man, 61 years of age, whom Dr. John Hynes had previously treated for carcinoma of the larynx. He was admitted in October 1957, developed a fistula and Dr. Hynes repaired it with a piece of homogenous aorta.

**DR. J. HYNES:** This man had a laryngectomy for cancer of the intrinsic larynx in August. His course was complicated by a short period of a pharyngeal fistula which healed before his discharge from the hospital. He went home, and was swallowing fairly well, but gradually developed progressive difficulty in swallowing. He was readmitted and studied. It proved impossible to pass a Levin tube at that time. X-ray examination showed a marked stricture of the cervical esophagus above which there was also a small diverticulum, apparently coming off the bottom of the pharynx. He was explored soon after admission. The stricture was located amid a lot of scar tissue and apparently was due to the previous pharyngeal fistula which had closed off at the skin level, but had left a chronic in-

flammatory mass with granulation, up against the wall of the esophagus where a marked stricture was present about the mid-cervical esophagus. The esophagus was freed as well as possible above and below this point. The stricture was incised longitudinally and sutured transversely. The tissues still showed some chronic inflammatory reaction and were rather poor stuff to anastomose. Within a few days, the closure broke down and he had a large esophageal fistula in his anterior mid-neck and unfortunately, because he had a mid-line incision, was spilling over into his permanent tracheostomy. The situation was serious in that he was aspirating his own secretions. Of course, he was being fed through a tube so that he wasn't getting food down his trachea, but he was getting

a terrific amount of mucus and saliva from his throat that would work into the trachea through the groove above his permanent tracheostomy tube, and he had severe coughing and expectoration. (Figure 2.)

Our next attempt was to repair the defect in the esophagus by using a piece of preserved aorta, reconstituted in saline, and suturing it into the defect. That was carried out approximately ten days after the first operation. Dr. Walter Moore kindly provided the graft which was sutured above and below to the open ends of the esophagus. The strip of tissue along the posterior wall which had been the site of the stricture was not removed but the esophageal graft was split longitudinally and sutured on each side of that.

At the end of the operation, the closure seemed to be adequate. There were two layers of very fine sutures placed, but within two or three days it was apparent that he had a second fistula. I think that in dissecting the upper end of the esophagus which was buried in very dense scar tissue near the base of his tongue, we probably opened into the diverticulum I mentioned and he had a salivary leak which bathed everything in mucus and saliva. He finally lost the upper end of his aortic graft. Whether the lower end is still there or not, I cannot tell, because meanwhile the skin had healed over and it is out of sight. Whether it has gradually disintegrated or not, I'm not sure, but he now has reached the point where he has a very small mid-line fistula high in the neck which leaks only a little. He is still on tube feedings, he has been home from the hospital a month or so, and I think the major problem is whether he has a sufficient lumen to prevent a recurrence of the stenosis. I can't give the answer to that.

He has gained weight on his tube feedings; he is comfortable, and he may yet have recurrences of the stenosis.



FIGURE 2

This barium swallow shows a diverticulum of the anterior lower portion of the pharynx. Just above the tracheostomy the stricture of the esophagus is noted.

## ASIATIC INFLUENZA COMPLICATED BY MYOCARDIAL INFARCTION REPORT OF A FATAL CASE

GEORGE J. BOINES, M.D.,\* EDWARD M. BOHAN, M.D.,\*\*

JASON L. CAMPBELL, M.D.\*\*\*

A white man, aged 54 years was admitted to St. Francis Hospital on September 25, 1957, with the history of having been ill at home for three days. His illness began with chills, fever, headache, dyspnea, abdominal cramps, a productive cough with expectoration of yellowish sputum, and general weakness. He treated himself for a cold until September 25, when his temperature rose to 103°, and he developed marked dyspnea and discomfort in his chest. He also noted that his right testicle was enlarged and tender, and he thought that this was related to his right renal calculus which had been diagnosed in 1952. His cough had been present for at least three or four weeks, but it was not serious enough to put him to bed.

On September 15, he had received 1 c.c. of monovalent Asian flu vaccine at his place of business.

His past medical history was negative

except for a small renal calculus in his right ureter which produced renal colic in 1952. He was treated conservatively, but the stone was not expelled. In 1950, he had a small calculus in his left ureter which he eventually expelled. He was allergic to aspirin. He was always overweight, averaging 225 to 240 pounds. His height was 68½ inches. He was immunized for typhoid-paratyphoid and small pox before sailing for Egypt and Greece earlier in 1957.

On admission, his systemic review was negative except for anorexia since the onset of the present illness. Blood pressure was 150/60, pulse 100, regular and strong. Temperature (rectally) was 103.4°F., dropped to normal on 9/28, and rose to 104.6° on 9/30, and gradually dropped to 100°F. Weight at this time was 225 pounds. Heart was normal without murmurs. There were moist rales, described as subcrepitant, at the right base, with generalized slightly diminished breath sounds. Liver and spleen were not palpated. There was no cyanosis. There was no ankle edema, nor was there any jaundice. The right testicle was swollen and tender.

\* Director, Department of Medicine, St. Francis Hospital. Attending Chief in Communicable Diseases and in Medicine, St. Francis Hospital.

\*\* Director of Research and Public Relations Committees, St. Francis Hospital. Attending Chief in Medicine and Director of Metabolic Clinic, St. Francis Hospital.

\*\*\* Assistant, Department of Medicine, St. Francis Hospital.

### Asiatic Influenza: Report of a Fatal Case

#### LABORATORY STUDIES

#### Insert I

BLOOD															
9/25 Mazzini negative															
	RBC	WBC	Hemoglobin % <sub>o</sub>	Gm.	Seg.	Lymph.	Hema- to- crit	Pro- thrombin	Sugar (2 h. P.C.)	B.U.N.	CO <sub>2</sub>	Chlorides	Na	K	Blood Culture
9/25	4,660,000	13,650	94	14.5	91	9	43.5		133	9.8					
10/1										24.9	48 vol. % <sub>o</sub>	625	152	4.1	Sterile
10/2	3,510,000	26,300	91	14	96	4									
10/4		18,500	85	14											
10/10	4,590,000	15,900	91	14	85	15			142						
10/13								81% <sub>o</sub>							
10/18	4,880,000	18,400	98	15	91	9				42.5					

## Asiatic Influenza: Report of a Fatal Case

## Insert II

## SPINAL FLUID

	Wassermann	WBC	Polys.	Lymphs.	Total Protein	Globulin	Chlorides	Sugar	Smear	Culture	Color	Pressure
10/7	Negative	5	3	2	79	0	775	92	0	0	clear	normal

11/16/57

**AGGLUTINATION TESTS FOR VIRUSES**

Reported by Dr. Klaus Hummeler, Virus Diagnostic Laboratory, Reference Laboratory for the Department of Health, Commonwealth of Pennsylvania, 1740 Bainbridge St., Philadelphia, Pa.

Lymphocytic Choriomeningitis	Mumps	Polio I	Polio II	Polio III	Influenza B	Q Fever	Psittacosis	Soluble Influenza A	Asian Influenza	L3 Strain	Adeno
0	0	0	0	0	0	0	0	1:32+	1:32+	1:32+	0

**URINE**

	Reaction	Sp.Gr.	Albumen	Sugar	WBC	Culture
9/25	acid	1.016	trace	0	many	
10/1	acid	1.010	0	0	15-20	
10/2	sl.alkaline	1.015	1+	0		Proteus vulgaris

**THROAT CULTURE**

10/3	1. Staphylococcus aureus	2. Streptococcus anhemolyticus	3. Diplococci
------	--------------------------	--------------------------------	---------------

**COMPLETE BLOOD VOLUME DETERMINATION** 10/2

1. Total Plasma Volume	- Deficit	0
2. " Blood "	"	0
3. " Plasma protein	"	0
4. " Circulating hemoglobin	"	31
5. Red cell volume	"	214 c.c.

## Asiatic Influenza: Report of a Fatal Case

## Insert III

**ELECTROCARDIOGRAPHIC TRACINGS**

	Rhythm	Rate	PR	Q	ST	Axis	Opinion
9/28	Sinus	74	.18	0	Normal	Normal	Within normal limits. QT interval is short. No evidence of hypokalemia.
10/12	Sinus	100	.24	0	Depressed		Distinctly abnormal septal damage
10/14	Sinus	110	.22	0	Depressed		Abnormal as on 10/12.

**X-RAY STUDIES**

9/25	Viral pneumonitis, right lower lobe
9/30	Parenchymal changes over right lower lobe
10/17	Hilar shadows are increased. No lung parenchymal changes. Heart within normal limits.

**PROGRESS NOTES**

- 9/25 No ankle edema. Temperature 103.6°F., pulse 128, respirations 40. Coughs Penicillin started.
- 9/26 Feels slightly better. Still weak and coughing. No jaundice.

- 9/28 Lungs are congested; moist rales throughout chest. There is impaired resonance over right base. Abdomen distended. He is confused and toxic. Some cardiac decompensation.

- 9/29 Right epididymitis is painful; however, patient feels better. Pneumonitis localized at right base. Poor appetite. Digitalis started.
- 9/30 General condition appeared improved in the morning. In the afternoon, he become somnolent and lethargic. No neck rigidity. No Babinski. BP 160/60. Pulse 100. Eyegrounds negative. Very restless. Complains of back pain. In the evening, the pneumonic process extended. Patient became extremely lethargic and was placed in oxygen. Temperature 104.6°F. after severe chill at evening.
- 10/ 1 Face dusky, pulse 72 regular; tongue dry, some neck rigidity, but no headache. Good urinary output, but blood urea is elevated to 24.9 mg. Right testicle is markedly swollen and tender. Gamma globulin (10 cc.) was given empirically for possible mumps orchitis. Temperature dropped to 101°F., pulse 80, respirations 24. Profuse perspiration present. Abdomen is distended.
- 10/ 2 Process in the right testicle has extended. May be responsible for the fever and abdominal distention due to deep lymphatic drainage. Perspiring.
- 10/ 3 General condition improved. Lungs clearing. Urinary output has been good.
- 10/ 4 Right orchitis is subsiding. Patient appears confused.
- 10/ 5 Appetite improved. Cheerful. Talking clearly.
- 10/ 7 Some neck rigidity; drowsiness appears of the encephalitic type. Spinal tap reveals normal spinal fluid except elevated total protein 79 mg. Still in oxygen tent.
- 10/ 9 Generally improved. Out of bed in wheelchair for short intervals.
- 10/10 Restless and drowsy. Good appetite.
- 10/12 EKG shows changes indicative of possible myocardial infarction. BP 170/60, pulse 80, irregular but strong. Temperature 99.2°F. Lungs

clear; no cardiac murmurs. Patient appears very pale and complains of extreme weakness and drowsiness. Still in oxygen.

- 10/14 BP 160/90, P.108, R.24. Coughs and expectorates small amounts of clear mucus. Still drowsy.
- 10/16 Pulse is slightly irregular, but strong. Patient is very tired.
- 10/17 Lethargic. Responds to questions. No cardiac murmurs; bradycardia plus extrasystoles. Lungs are clear. No nuchal rigidity. Abdomen is soft. Femoral pulses present. Patient is very restless.
- 10/18 Pulse very strong and regular; not confused. Out of bed. Nauseated and vomited. BP 180/98, pulse 94, respirations 24. General condition seems improved. At 11 P.M. BP 170/60, restless, poor color. Gasping for breath; expired 11:15 P.M.

*Cause of death:* 1. Myocardial infarction due to coronary thrombosis  
2. Viral pneumonitis — Asian influenza  
3. Acute epididymitis  
4. Right renal lithiasis with uremia and hypertension

*Therapy:* 1. Antibiotics  
2. Parenzyme I.M. for orchitis  
3. Digitalis  
4. Fluid therapy, vitamins, Prostigmine  
5. Oxygen  
6. Gamma globulin  
7. Tranquilizers

## DISCUSSION

### CLINICAL CHARACTERISTICS OF VIRUSES

Viruses are obligate intracellular parasites. The symptoms in viral infections are dependent on capillary damage which leads to increased permeability with protein leakage into the tissue and their sequelae.

Viruses are meningo-tropic, dermatropic, neurotropic, pneumotropic, viscerotropic and cardiotropic. Some have a preference for several tissues simultaneously or may be pantropic. Some agglutinate erythro-

cytes. The clumping of red cells by viruses is specifically inhibited by homologous immune sera. This leads to the practical use of agglutination inhibition tests. Viremia may occur.

Myocardial involvement may be brought about by the action of one or more viral agents, sometimes one activating the other. Antigen-antibody reaction may also occur in the heart, or secondary infection may be responsible for its damage.

Various studies show that the primary pathology of influenza virus infections is destruction of respiratory tract epithelium. Type A influenza virus was isolated by Smith, Andrews and Laidlaw in 1933. They infected ferrets with filtrates of secretions obtained from the nasopharynx of patients in the early stage of influenza. The English strains and the Puerto Rican strains now are known as influenza virus A.

A group of new strains appeared in 1946 and 1947 which were related to Group A, but did not cross protection with the A strain in man or animals. These strains are now called A prime.

Some cases have no respiratory infections. The onset is abrupt or sometimes preceded by mild malaise. Anorexia, fever, headache and injection or burning of the eyeballs are common. Mild sore throat and general aching may be common. The temperature varies from 101° to 104°. Exhaustion occurs. Usually neutropenia is present.

#### THE ASIAN INFLUENZA STRAIN

This is also known as influenza A — Asian strain (FE).

It was first identified by Dr. Maurice R. Hilleman from the Hong Kong epidemic in May 1957. The spread of this upper respiratory infection throughout the United States is now past, but it has left an impressive history in the annals of medicine. The average duration of symptoms is three to five days. October and November,<sup>1</sup> 1957, marked the highest peak in the incidence of pneumonia and influenza all over the United States. The Asian influenza virus

was isolated from numerous patients during this period.

In a study of twenty-three deaths, Herrmann et al<sup>2</sup> reported the following symptoms predominating in the acute stage of this disease: headache, cough, sneezing, anorexia, chills, fever, chest pain, hemoptysis, "cold", rhinorrhea, malaise, crepitant and musical rales, dyspnea, sore throat, lethargy and weakness, aches in bones and joints. The usual organisms cultured from the respiratory tract secretions were Friedlander's bacilli, alpha streptococci, pure culture of *H. influenza*, and pneumococci and or micrococci (staphylococci) from 17 of the cases (Results of the coagulase test were positive.)

The pathology noted in posted material included severe laryngitis with heavy exudate, bronchiolitis, pneumonia with edema, atelectasis and acute focal emphysema, encephalomalacia, minimal meningitis, and cerebral edema. The findings in the lungs which are consistent with the pneumonia classically attributable to influenza are as follows: "Grossly the lungs were externally patchy, dark blue gray, and firm. On section, the parenchyma was excessively moist; a gray brown watery fluid ran out freely, and the parenchyma was dark red and firm. Microscopically, there was extensive filling of the alveoli with pink fibrin, and, in areas, there was a hyaline membrane formation lining some of the alveolar walls."

X-ray findings were usually negative even if there were clinical lung findings.

In a study of 50 fatal cases by Davenport<sup>3</sup>, it was noted that approximately one-half showed evidence of staphylococcal pneumonia. The others were either overwhelming infections by organisms readily responding to early conventional therapy, or to less intense infections in persons with chronic disease. A small number of cases were associated with infections in the last trimester of pregnancy. Some of the pneumonias were recorded as hemorrhagic. The pneumonia cases responded well to antibiotics.

Our case outlined above had all of the symptomatology typical of influenza. How-

ever, he also had the epididymitis and orchitis, mild meningeal symptoms, and drowsiness, restlessness, weakness which accompany encephalitis. These complications are also seen with Q fever and mumps meningoencephalitis. The virus complement fixation tests were negative for these however, but the tests were positive for Asian influenza. The pneumonitis cleared up clinically and by X-ray evidence. Our patient was apparently improving when he suddenly expired: a cardiac failure death.

The therapy consisted of antibiotics, digitalization and supportive measures. Gamma globulin (10 c.c.) was given intramuscularly when it appeared that he might have had a mumps meningoencephalitis. His general condition improved temporarily after the gamma globulin.

#### IMMUNIZATION BY INFLUENZA VACCINE

The effectiveness of the Asian influenza vaccine in producing immunity against this disease was demonstrated by Bell et al,<sup>4</sup> who induced Asian influenza artificially in vaccinated and unvaccinated volunteers. The vaccinated group did not develop fever as the controls, and the symptoms were more serious in the placebo group after challenge with the virus. Of the unvaccinated group 78% developed influenza as compared with 44% of the vaccinated group after they were infected with the virus.

Antibody increase is not expected until ten days after vaccination. The antibodies appeared 17 to 19 days after the challenge with the virus. The fever and other symptoms appeared 33 to 44 hours after the challenge. In several studies with controls, the vaccine was found to be 42% to 61% effective with the 200 CCA vaccine and 67% effective when the 400 CCA unit vaccine was used. The more concentrated the vaccine, the greater the percentage of effectiveness. The vaccine effectiveness lasts about six months.

#### DOSAGE OF VACCINE

There are two influenza vaccines available at the present time. The monovalent

Asian influenza vaccine is made in 200 and 400 chicken cell agglutinating units per c.c. and the polyvalent influenza vaccine contains influenza A, several strains, B, one or more strains, and the Asian influenza strain.

The following dosages of Asian influenza vaccine are recommended:<sup>5</sup>

1. New patients 1.0 c.c. of the 400 CCA.
2. Those who had received 0.1 c.c. intracutaneously or 1.0 c.c. subcutaneously of the 200 CCA unit vaccine and for those individuals of high risk (pregnant women and persons with chronic cardiac and respiratory conditions), a second 1.0 c.c. subcutaneous injection of 200 CCA vaccine.
3. If the 400 CCA unit strength vaccine is available, 0.5 c.c. should be given and repeated two weeks later.
4. Children under 5 years of age should be given 0.1 c.c. intracutaneously or subcutaneously and repeated after one to two weeks.  
Children from 5 to 12 years of age, 0.5 c.c. subcutaneously, repeated after an interval of one to two weeks.  
Children over 13 should receive the adult dosage.
5. A new polyvalent influenza vaccine is now in production containing:

Influenza A Asian strain	200 CCA
" A <sup>1</sup>	100 CCA
" A	100 CCA
" B	100 CCA

This vaccine is more effective. It is given 1.0 c.c. subcutaneously and repeated in two weeks. The vaccine should not be given to individuals who have a history of allergy to eggs. The reactions encountered otherwise are minimal and occur during the first 24 hours after administration.

#### VIRAL DIAGNOSIS OF ASIAN INFLUENZA

The etiologic diagnosis of influenza depends on virus isolations or antigenic findings<sup>6</sup> since many other infectious agents produce clinical syndromes similar to influenza. The laboratory methods in identifying viruses at the present time are:

1. Electron microscope
2. Tissue culture in cells from monkey kidney, HeLa, or human embryonated
3. Hemagglutination-inhibition by antibodies
4. Antibody neutralization of tissue cultures
5. Complement fixation
6. Cultivation of viruses in fertile eggs

An early (24-48 hour) specific diagnosis of influenza or any other viral infection is imperative in our everyday medical practice, in the home and in the hospital. It is important for us to know early in the course of the disease whether we are dealing with a virus or a bacterial infection. Even though a specific antibiotic agent may not be available today, it may become available tomorrow. On the other hand, it is of the utmost importance to the patient to treat his bacterial infection energetically with the indicated antibiotics, whereas, the viral infection requires more conservative therapy.

Improved techniques in growing viruses<sup>7,8</sup> in tissue cultures have made possible the early isolation of previously unknown viruses from human upper respiratory passages and the intestinal tract. The performance of various serological tests for antibodies in paired sera from cases have simplified procedures for further placing these viruses into their specific type. Thus, poliomyelitis, Coxsackie, herpes simplex, influenza, mumps, measles, varicella, adeno-

virus or ECHO viruses are identified. Many of these viruses produce typical cytopathogenic changes in the tissue cultures within 15 to 24 hours, and a virologist is able to identify the viral infections group by simply placing the test tube containing the cultured tissue cells under the microscope for examination. With such simple methods of identifying viruses, we can easily determine, for example, whether aseptic meningitis is caused by Coxsackie, ECHO, or non-paralytic poliomyelitis viruses.

In a previous communication,<sup>10</sup> we pointed out that as physicians of Delaware, we have thus far been deprived of early virological diagnoses in our patients. Now is the time to emphasize to all concerned that viruses are here to stay and that the most advanced diagnostic laboratory facilities in this field should be made available to our patients.

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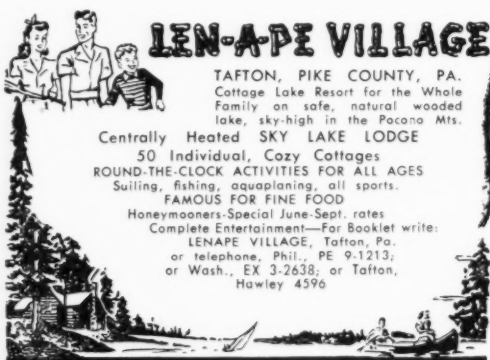
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## CHRONIC MYELOID LEUKEMIA: DIAGNOSIS AND TREATMENT

JAMES M. HOFFORD, M.D.\*

The following case presents an interesting problem in the differential diagnosis between chronic myeloid leukemia, myelophthistic anemia, and leukemoid reaction. While the diagnosis is simple upon reviewing the data, it actually was not made until the final bone marrow biopsy was obtained.

### CASE REPORT

The patient was a 72 year old white woman who complained of soreness in the low back with radiation into each buttock; this had been present for one year prior to admission. One week before admission the tenderness on the left side had increased and was aggravated by walking. She also complained of weakness, dyspnea, edema of the legs, and heaviness of the abdomen.

The patient had been admitted to this hospital in 1949, at which time a vaginal hysterectomy and posterior colporrhaphy were done. She was readmitted in 1954 because of coronary thrombosis and hypertensive cardiovascular disease. Review of the old records did not reveal any evidence of an enlarged abdominal organ during those years.

There were no significant family illnesses. She came to the hospital from a home for the aged, where she had been living a comfortable existence, maintaining an optimistic and happy outlook.

**Physical examination:** The pulse was approximately 120 and irregular due to auricular fibrillation. There were a few small discreet nodes in the left supraclavicular region. The breath sounds were distant and there were coarse and fine rales over both lung fields posteriorly, especially

at the bases. The heart sounds were distant and the apex beat was almost to the anterior axillary line. There was a grade 1 systolic murmur. The liver edge was sharp, smooth and non-tender and was located halfway between the costal margin and the iliac crest. The spleen was enlarged to the midline below the umbilicus; it was firm, smooth and non-tender. Pelvic and rectal examinations were negative; the uterus was absent. There was 3+ pitting edema of the ankles and sacrum.

### Laboratory studies:

Admission blood count—(11-20-57)

Hb.—58%  
Red blood cells—3,010,000  
White blood cells—67,300  
Neutrophils .....54%  
Bands .....28%  
Eosinophils .....2%  
Lymphocytes .....5%  
Myelocytes .....4%  
Metamyelocytes .....7%  
Normoblasts .....2%

12-4-57 Reticulocytes—1.2%  
Platelets 238.26 in 1000

12-9-57 Hb.—64%  
Hematocrit—38  
Reticulocytes—3.2%  
White blood cells—61,400

Mazzini test—negative. Feasting (2 hour p.c.) blood glucose—115 mg.%. Blood urea nitrogen—12.5 mg.%. Urinalysis—negative except for trace of albumin. Direct Coomb's Test—negative. Indirect van de Bergh—0.8 mg.%. Total protein—5.2 Gm.%. Albumin—3.9 Gm.%. Globulin—1.3 Gm.%. Alkaline phosphatase—6.6 Bodansky units. Prothrombin time—81.9%.

Electrocardiogram—auricular fibrillation with premature ventricular complexes.

\* Assistant, Department of Medicine, St. Francis Hospital, Wilmington, Delaware.

**X-ray studies:**

Chest—cardiac enlargement, left ventricular; arterio-sclerosis of aorta; pulmonary congestion.

Upper gastrointestinal series—normal except two duodenal diverticuli.

Barium enema — diverticulosis; downward displacement of splenic flexure of colon by mass in left upper abdominal quadrant.

Intravenous urogram—poor renal function.

Sternal bone marrow aspiration (11-30-57)—scanty specimen; reduced cellularity, chiefly myeloid elements; few nucleated red cells; many platelets.

Bone marrow biopsy (12-19-57) — marked hyperplasia; no fat spaces; most cells of myeloid series; goodly number of red cell elements and megakaryocytes. Impression: Chronic myeloid leukemia.

**DISCUSSION**

The initial bone marrow aspiration showed a relative hypoplasia of the granulocytic series. The diagnosis of a myelophthitic anemia had apparently been made. As can be seen, this points out the limitations of a single bone marrow aspiration. A myelophthitic anemia is one in which the bone marrow is hypoplastic, fibrotic, or sclerotic. There is a striking splenomegaly. There are immature white and red blood cells in the peripheral smear, and there is myeloid metaplasia of the spleen and liver.

A myelophthitic anemia may be caused by metastatic carcinoma in the bone marrow, multiple myeloma, myelofibrosis, "marble-bone" disease or osteopetrosis, Hodgkin's disease, Gaucher's disease, Niemann-Pick's disease, Schuller-Christian's disease, osteitis deformans, osteitis fibrosa cystica, osteomalacia, osteogenesis imperfecta cystica, poisoning by fluorine, phosphorus, estrogens, charcoal, anterior pituitary extract, parathyroid extract, and irradiated ergosterol.<sup>1</sup>

With the presumptive diagnosis of a myelophthitic anemia in mind, treatment

was discussed. Radioactive phosphorus, P32, which is incorporated into the nucleoprotein of cells, inhibits the growth of cells which are multiplying at the fastest rate. In cases of leukemia, this therapeutic agent is found concentrated in the liver, spleen, kidneys, and bone marrow.<sup>1</sup> It seemed logical to assume that the agent would interfere with the extramedullary hematopoiesis in the liver and spleen, and thus thwart the body's attempt to maintain a normal red blood cell count.

Radioactive phosphorus is in reality a form of radiation treatment. Hickling<sup>2</sup> reported nine patients with myelosclerosis, seven of whom had x-ray treatment. Of these seven, two patients received no benefit. These two patients had not initially had an increase of the white blood count. The remaining five patients benefited from x-ray treatment. All five had an increase in the circulating white blood cell count from 20,000 to 70,000 before x-ray. The spleen usually decreased in size following these treatments.

Hickling also reported that he had found in the literature 14 cases who had had splenectomy. Four of these patients died within three months, and four died between ten months and six and one half years. Of the six who survived splenectomy, two lived a short time, and four lived one to five years. There was no striking improvement in any of the patients who had received splenectomy, except one who was reported alive several months after the operation and who did not require the frequent transfusions he had prior to operation.

Videbaek<sup>3</sup> mentions that patients with marked leukocytosis and numerous immature leukocytes in the blood are believed to respond to x-irradiation of the spleen despite the osteomyelofibrosis. He mentions the common features of cases where splenectomy has had favorable effects. These features are slight leukocytosis, small numbers of immature myeloid cells in the peripheral blood, pronounced thrombocytopenia, and chronic splenic hemolysis. He mentions that Loeb, in 1953, splenectomized two patients successfully. In Loeb's opinion, a favorable response to cortisone

treatment indicates the need for splenectomy. Loeb states that thrombocytopenia and hemolysis in myelofibrosis are operative indications.

Videbaek reported three patients. The first had a thrombocytopenia and myelofibrosis. This patient improved decisively with splenectomy and had been maintained two years after operation. The second patient had a reticulosis of the bone marrow and anemia. This patient did well, but developed chronic lymphogenous leukemia. Patient number three was initially thought to have chronic myelogenous leukemia, but later a diagnosis of myelosclerosis was made. The patient was treated initially with Myleran and maintained for two years. The spleen decreased to normal size.

The bone marrow biopsy which was finally obtained suggested the diagnosis of chronic myelogenous leukemia in the present case.

A leukemoid blood picture was ruled out on the basis of exclusion, as can be seen from the accompanying data. The differentiation between leukemoid reactions and true leukemia is often extremely difficult.

Leukemoid reactions have been observed in association with infections: bacterial, spirochetal, viral, and protozoal. In pneumonia with empyema, in pneumococcal endocarditis, meningococcal meningitis, and diphtheria, white blood counts of approximately 70,000 have been recorded. Bubonic plague and septicemia have produced white counts of 100,000. Marked leukocytosis and lymphocytosis have been described in whooping cough (176,000), chicken-pox (81,000), congenital syphilis (105,000), infectious mononucleosis (63,000), and infectious lymphocytosis (147,000).

Tuberculosis has frequently been described as producing a leukemoid blood picture, with white blood counts as high as 156,000.

Intoxication produced by eclampsia, severe burns, mustard gas poisoning, and the use of 33% mercury ointment, have produced leukemoid reactions with blood counts ranging between 69,000 and 100,000.

Malignancy has produced leukemoid blood pictures without bone metastasis. Carcinoma of the colon may show metastasis to the spleen. Carcinoma of the stomach with lymphoid reaction, carcinoma of the lung, adrenal and renal carcinoma with widespread metastasis may show necrosis.

Severe hemorrhage and sudden hemolysis of blood may produce a leukocytosis of 106,000.

In pernicious anemia, a white blood count of 69,000 during remission following liver therapy has been reported.<sup>1</sup>

In chronic myeloid leukemia, the differential count of the bone marrow is similar to that of the peripheral blood. The cells are at a slightly less mature level than in the circulating blood. Some observers have described an increased number of megakaryocytes.

In chronic myeloid leukemia, the blood platelets are usually normal or slightly increased in number. There is usually in chronic myeloid leukemia a well marked leukocytosis with counts ranging from 100,000 to 800,000 per cu. mm. A count of 200,000 to 400,000 is frequently found at the time the disease is first discovered.

Lower counts may be found when the disease is more advanced.<sup>1</sup>

It was decided that this patient had chronic myeloid leukemia, and the therapeutic agent would be P32, radioactive phosphorus.

#### SUMMARY

1. A case of chronic myeloid leukemia is presented.
2. The diagnosis and treatment of myelophthisic anemias is discussed.
3. The importance and difficulty of differentiating leukemoid reactions from chronic myeloid leukemia is discussed.

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## THE DOCTOR AND THE TRUST DEPARTMENT\*

RODMAN WARD\*\*

I have noticed that it is common for a member of the medical profession to have his home telephone number unlisted or at least disguised by being in the name of his wife, and that he finds it necessary to hide out or leave town in order to enjoy a few hours of personal freedom. These practices are not usual for people of other occupations and are not done without reason.

The reason, I believe, is that the doctor more than any other professional man and very much more than the man in business, is subject to frequent, imperative and interruptive demands upon his time.

Also it is peculiarly true of the medical profession that you cannot multiply the number of earning hours in the day, as practically every one else can, by the use of employees and substitutes and by delegation to subordinates. Even a lawyer will have a junior do his research for him, and the hours spent by the junior in digging out the law of the problem will be condensed into a neat memorandum for his employer's benefit.

In a manner of speaking, a doctor's stock in trade is his time. Time available for his practice is, to him, money in a strict sense.

By the way, as a group I understand you are doing pretty well in spite of this special difficulty. I have noticed that the American Bar Association has commiserated with its members in pointing out that incomes of doctors have lately increased at a rate faster than have the incomes of lawyers.

The only non-professional subjects you have scheduled for this session of your Society—taxes, investments and trust departments—all would indicate that you

have successfully converted some of your past time into dollars that have stayed with you and you feel the need of giving them some attention.

I take it, therefore, that you would be interested in how proper attention can be given to your accumulated earnings and to future accumulations so that they will not be lost, will even increase, all at a minimum expenditure of the valuable commodity, the hours in your day. The availability of this kind of service is, I think, the special interest of the doctor, as doctor, in the trust department.

First I would like to explain something of the nature of a trust department, and from that, go on and mention its services best adapted to meet the demands of your special situation.

A trust cannot exist without the involvement of something of value. This is held — is legally speaking *owned* — by the trustee, but only for the benefit of one or more other persons, the beneficiaries. It is an old institution which got along for years without there being any trust companies. However, with the development of the popularity of investments in bonds and shares of stocks and the increased respectability of these forms of property which occurred during the last century as a shift from concentration of investment of accumulated moneys in real estate, a need developed for a professional type of trustee, as opposed to the family friend or the attorney who had commonly filled the bill up to then. The new type of property needed more continuous attention. Also it was more subject to the risks of dishonesty in the individual who, holding legal title to it, sometimes found it easier to convert to his own use than real estate would have been.

Out of this need, developed the corpora-

\* Presented at the Annual Meeting, Medical Society of Delaware, Wilmington, 1957.

\*\* Vice President and Trust Officer, Equitable-Security Trust Company, Wilmington.

tion as a trustee. From the beginning corporate trustees were hybrid affairs. They were also engaged in banking, as are the trust companies in Wilmington, or in the life insurance business, as was the Provident Life Insurance and Trust Company, later split into the Provident Trust Company and the Provident Mutual Life Insurance Company in Philadelphia.

For some reason corporate trusteeship fared better under the same roof with a bank, and now you find an almost but not quite complete concentration of corporate trust business in this country in banking institutions. In England there is a forced separation between banking and trust business.

However, in all well developed and properly run banking institutions which engage in trust business you will find that the trust department is largely a separate entity. This is proper because the nature of trust business and the laws which govern it are as different from the nature of banking business and its laws as is green from red. For example, it is common and proper that the Board of Directors of a trust company has its Executive Committee which attends to the affairs of its banking department, and its Trust Committee which interests itself only with the affairs of the trust department. The officers and other personnel who attend to the business of one department are quite separated from the business of the other department. It is even common that the trust department investments are kept in a completely separate vault under controls that are entirely separate from the controls over the vault of the banking department.

Because of this split personality which has developed in this country, we speak of the trust department. If we were in England we would speak of the trustee company.

Very quickly after the development of the corporation as trustee, the unique combination of qualities and facilities which it offered were found to be desirable for more activities than the strictly fiduciary—that is trusteeship, guardianship and executor-

ship. The trust department was found also to be useful for various forms of agency.

The first of these qualities is financial responsibility. The capital, surplus and undivided profits of the trust institution are a fund available to guarantee this, and in a company of relatively good size they amount to millions of dollars. Banks are subject to so many requirements of reports, of examination by public agencies, of audits and of publication of statements that you can rely on this guarantee fund.

Another of these is that a trust department is at work, full time, in attending to the business of its customers. There is no question of whether its own affairs should be attended to first. It has no business other than the business of its customers. A well-organized trust department has a considerable staff largely made up of people who specialize in one facet or another of its activities. It has people who are constantly watching the security markets and the prospects of the general economy and the economy of individual companies. It has real estate men, and tax men, and people whose duty it is to maintain familiarity with other kinds of law affecting the business of its customers in which it is engaged. The trust department is organized in depth, also, so that illness and vacations do not interfere with its operation. A trust department is also organized in such a way that one customer need not, in ordinary course, run from one man to another, but may limit his contacts to one or two officers with whom he becomes familiar and who acquire familiarity with his wishes and objectives and general situation.

What are the kinds of services which are offered and which may be useful to the doctor? I shall start with those in which you might be interested for yourself, now, as a living person and work into the future.

The simplest, perhaps, is what is called a custody account. This is for the man with investments who wants someone to hold them for him, to collect the income, to watch out for and to notify him when any securities are called for payment or

when rights are issued and the like, and to keep records for him. The custodian service does not provide recommendations for investment changes. I mention it without the idea that it would be widely attractive to this group.

One step beyond this, and of a nature that should be useful for many a doctor, is what is variously called a managing agency account, an investment advisory account, or simply an agency. This embraces all of the services provided in a custody account, plus—and it should be a big plus for the busy professional—regular investment review and recommendation. Such an account is not a trust because the investments remain yours, completely and in all respects, and you retain full control over them, but you are provided with the advice of investment specialists on which to base your decisions. A doctor, whose professional income is limited by the hours of the day available for his practice, obtains through such a relationship with the trust department, an accurate bookkeeper, a responsible custodian, and an experienced and loyal adviser as to his investments. I say "loyal" because a trust department has nothing to sell to him except advice, which it believes is for his best interests. The trust institution does not and must not sell to him securities which it owns, or make side commissions or secret profits out of its business relationships. It maintains itself in an independent position, in a position where its interests do not conflict with those of its customers.

Here, I want to interrupt this catalogue of trust department services to comment on the nature of the investment advice you will get, or perhaps more importantly, to point out what you won't get. Trust investment officers are a *relatively* conservative bunch. They are trained in an atmosphere where "speculation" is a dirty word insofar as their business is concerned. They will not hold out the promise of quick profit through short-term stock market price swings. They will not even say to you "Here is a stock in a little company just started in a new industry which has a good chance of really going places. You might

increase your money in it by ten times over a few years."

On the other hand they won't say that a government bond is the only safe investment.

They will first ask you what are your general objectives as between a high but sound rate of income return and less income but greater hopes for building up value over a longish period. They will probably ask if part of your fund should be kept in backlog investments to meet some anticipated need for cash, or to meet an emergency which may arise and is not provided for otherwise.

Then, to fit your situation, they may suggest part of your funds be in tax-free bonds, or quality corporation bonds, or preferred stocks, and that the balance—maybe all—be put in common stocks of selected, well-established corporations where the management has proved itself and for which the future holds real promise. They will recommend a reasonable amount of diversification because they will admit they don't pretend to *know* that ABC Company is going to do well, or that XYZ Corporation is positively going to do better.

On what do they base their thinking? The situation in my own company is not atypical even though we think it is as good or better than the next. Of course we subscribe to standard services that provide information and to financial journals that include not only news but comment and recommendation. Then we pay a good big annual fee to an organization that concentrates on study of the future economy of different industries and of the country as a whole, and also makes investment recommendations to us, with reasons. When we wish, we talk to them about this or that problem or situation.

In addition, our investment officers are in touch with the investment officers in the larger banks in Philadelphia, New York or Boston. They make trips, for example, to New York to visit several banks and spend a day gathering the consensus of reliable and informed opinion, not so much from dealers in investments as from others in the

same business as is ours. We also maintain good relations with a number of brokerage houses because quite a few of these have extremely capable researchers and analysts on their staffs, and provide worthwhile ideas.

These are the sources of the grist for our mill, for we must make our own decisions affecting our own customers, informed decisions which are the result of the group judgment of our investment officers and committees based on information and opinion largely gathered elsewhere.

After the managing agency account in the catalogue of trust department services suited to doctors, comes the *inter vivos* or living trust and here I can only touch on a very complex subject. It has been said that the purposes and forms of trusts are "as unlimited as the imagination of lawyers."

A trust may be established and governed by the will of a dead man or by a written agreement between a living man and the trustee. This latter is the living trust, and it is effective during the life of its creator and usually continues after his death. The creator may keep as much control over it as he wishes, or he may completely divest himself of any control or even of any interest in the property which is subject to the trust. The trust may be revocable—that is it may be cancelled at the will of its creator—or it may be a permanent and irrevocable disposition.

As I said earlier, a trustee is, legally speaking, the owner of the trust property. He is, however, bound not only by the provisions of the trust agreement or of the will in question, but also by an extensive and very special body of law in order to assure that he manages the trust, not in any way for his own benefit but only to the advantage of the beneficiaries—the people who are intended to benefit by the arrangement.

Beyond this law, there is a code of ethics. And I quote with some pride what has recently been written by a justice of the Supreme Court of Oregon, which is: "I assert with confidence that the standards of ethics which trust officers have adopted and

by which they are actually bound are higher than those which bind any other group in the business world."

The living trust is usually adopted—in the place of an agency account—in order to obtain something more than mere help in investments. There are definite tax advantages available under some circumstances. Through a trust, entirely secret arrangements may be made for the benefit of others. As I said before, the creator of a trust may relieve himself completely of what he may consider the nuisance of having to make investment decisions. He may have decided that those fellows know a lot more about it than he does and that he will leave it up to them entirely—for the time being at least.

The agency and the living trust are the two pieces of machinery by which the doctor can obtain from a trust company some or even complete relief from having to spend his valuable time with the care of his investments.

Through one of these, there may be obtained useful psychological assistance toward a successful accumulation program. It is very hard to save under current conditions. Just as a program of paying life insurance premiums is a help along these lines, the doctor may promise himself and tell the trust company that he intends to make periodic additions to his trust or agency account and find he has put himself under sufficient compulsion so that he will follow through with his intentions. He may find that the slight formality required for the withdrawal of money from such an account is just enough to make him think twice—and not spend it.

Under my theory that it is typical both that the medical man lacks time for management of his non-professional affairs and that he has income far above the average, not unusually with some excess which he puts aside or at least wants to put aside in investments, it is not illogical to say that he will find in the trust department of his bank services, in the form of agency or living trust facilities, which are suited to his peculiar needs.

These are of course very far from every kind of way in which trust departments will serve to the advantage of you and your family. You have heard of estate planning. Undoubtedly many of you have received the benefit of it from one source or another. To those and to the others, I would say that too often and too much the estate planner limits himself to statistics about your family, your income and your resources, puts them down on paper and makes rather stereotyped recommendations which will result in certain tax savings and in a sale of the goods or services which the particular estate planner has for sale. There is a lot of value even in such a review of your affairs. But more value can be had if the man you consult will do a little prying into the more personal side of your family and if you will disclose to him your special family worries, the things that make your family different from another, so that the suggestions can be really tailored to you and not simply taken off the shelf as size 42, short.

I am prejudiced, I admit, but I do think that you have a somewhat better chance of this result if you go to the trust department of your bank for a discussion and for suggestions for arrangements you can make while you live which will leave things easier for your wife and children after you die. I *know* that few people have greater experience with human problems, which are sometimes really serious and at other times not so serious as they seem, than does a trust officer. I think this tends to make human values relatively more important to him than they may be to a man in another business.

These human values are important when it comes to a discussion about what you should say in your will. Technical proficiency on the part of the person you consult with is essential but by itself is not enough.

In any event the trust department offers its services, without charge, for discussions leading to recommendations, for the manner in which you may direct the disposition of your property at your death in order to

preserve as much as possible from erosion by taxes and expenses, in order to assure that what you leave will do what you want it to, and in order to provide whatever degree of protection you think members of your family may need. That is estate planning.

The man in the trust department talks to you with a background of experience of the kind that counts—of experience gained from having seen good plans and bad plans in operation over the years.

I could go on with an argument in favor of the bank or trust company as your executor. And I could go with some explanation of the testamentary trust—that is the trust which is established under the terms of your will—and how taxes can be saved by this or that provision. I could talk of the advantages of an insurance trust. The subjects are many and the talking could be endless.

Such things, however, are a little beyond my subject as I see it. I have wanted to give you some appreciation of what trust departments are and how they have developed in answer to a public demand for an institution of financial reliability and integrity, equipped with the technical abilities required for the successful management of invested money in our complex civilization, and yet an institution which has the personal touch—which will handle your money as your money and not as one small part of one vast fund. No one knows to what extent the collective trust departments of this country have been called on for service, and no one knows the total of the values in their care. The confidentiality of the business forbids the collection and publication of such figures. We only know in a general way that they add up to many billions of dollars—which is some indication of how successfully this institution is meeting the demand that brought it into existence.

And, more importantly, I have tried to suggest that the doctor particularly—the man who can't hire people to do his professional work for him—may find in a trust department the answer to some of his special problems.

## + Editorials +

### LOVE

When 450 townspeople gather to honor a family doctor, it is obvious that the motive is love—their love for the doctor. Less obvious, but equally important, is the doctor's love for the people; a love demonstrated by years of devoted service.

It is not necessary, however, for a physician to complete 45 years of service before being loved by his patients. Recently, during a heavy snowstorm, a young physician of Delaware walked quite a distance through snowdrifts to see a young child patient even

after being told by the parents that it would be all right to wait until the next day when a path would be cleared.

These are but two examples of the love physicians have for their patients; obviously, many others act likewise without formal recognition.

Congratulations, Dr. Chipman. We, your colleagues, are proud of you and the manner in which you practice the principles for which we all stand.

### PROTECTION FOR THE HIGH SCHOOL ATHLETE

There have been an unnecessarily high number of injuries and deaths resulting from high school sports; to curb this rate is a community challenge. Physicians can and should provide much needed local leadership by working with school officials, coaches, parent-teacher groups and the dental association to develop adequate school health and safety programs for sports participants. One practical method is the sponsoring of high school sports injury conferences; this is discussed in a new American Medical Association pamphlet. The

purpose of these conferences is to instruct coaches, athletic directors and team physicians on the early recognition of injuries, appropriate first aid measures and the prompt referral of injured players for medical or dental care. Entitled "*Protecting the Health of the High School Athlete*", the booklet was prepared under the auspices of the AMA's Committee on Injury in Sports. Further information and copies of the booklet may be obtained from the AMA's Bureau of Health Education.

### REACTION TO ANTIBIOTICS

In 1952 and 1953 the Food and Drug Administration made a survey of reactions to antibiotics in 11 cities and found 88 cases of anaphylactoid shock, 25 of which were fatal. These were all unreported cases and the drug involved in each case was penicillin, the broad spectrum antibiotics apparently being benign.

A second survey in 1956 revealed 733 anaphylactoid reactions of which 72 died, approximately 10%. These reactions followed the parenteral use of the drug; there were 49 reactions following its oral administration, none of which was fatal.

Study of the author's data reveals some interesting facts.

Penicillin is a highly antigenic drug of which several hundred tons are prescribed annually. With time there will be an increasing number of allergic reactions. Unfortunately, the size of the dose is unimportant, many patients having been "triggered" into severe anaphylactoid shock by minute amounts of the drug.

One patient went into shock following the use of a syringe contaminated with penicillin. Another was that of a woman who prepared a penicillin tablet in a sugar solution for her grandchild; she tasted the solution to determine whether it was sweet enough and quickly went into shock. A third went

into shock immediately following application of penicillin ointment to a skin abrasion. These are merely a few of many instances cited.

It is the responsibility of the medical profession to take seriously this warning and to do all things possible to combat this danger. The first, and obvious, step is to re-evaluate our indications for penicillin therapy and to use the drug only when there exists a definite and valid indication for its use. Such a simple step would do much toward eliminating the occurrence of these serious mishaps.

## OPHTHALMOLOGIST - OPTICIAN - OPTOMETRIST

There is a lawsuit pending in Delaware brought by the State Optometric Society and the State Board of Optometry against a Delaware optician. It might be well at this time to get a clear definition of these terms — ophthalmologist, optician, and optometrist.

An *ophthalmologist*, sometimes called an *oculist*, is a doctor of medicine who specializes in the care of the eye and all the related structures. He diagnoses and treats defects of focus, disorders of function, and all other diseases of the eye, prescribing whatever is required, including glasses. As a member of a medical team, he frequently is concerned with diseases of other systems of the body or general diseases which manifest themselves in the eye, such as diabetes, toxemia of pregnancy, cancer, multiple sclerosis, tuberculosis, high blood pressure, and heart disease. *Ophthalmology* is a branch

of medicine and the *ophthalmologist* is an eye physician and usually an eye surgeon.

An *optician* is a skilled technician who supplies and fits glasses on the prescription of a physician. He is trained to make the necessary facial measurements; to make the glasses or other appliances; and to adapt them to the patient. He supplies glasses or other appliances only on the doctor's authorization.

An *optometrist* is a licensed person who has met certain legal and educational requirements and is permitted by the state to engage in the practice of optometry. He is not a physician or a doctor of medicine. He measures the focus of the eye for glasses. He is neither qualified nor permitted to use drugs for these tests or for any other purpose. He is not qualified or permitted to diagnose or treat disease of the eye. He may supply glasses on his own prescription. By law he is a limited practitioner.

## WILLIAM THOMAS CHIPMAN, M.D.

*On February 24, 1958 about 450 citizens of Harrington honored Doctor Chipman after 45 years of the practice of medicine. The following introduction was presented by the Mayor of Harrington, Dr. Hewitt W. Smith, past-President of the Medical Society of Delaware, Dr. Chipman's colleague.*

Mr. Chairman, honored guest of the evening, distinguished guests at the speakers' table, ladies and gentlemen:

When your program chairman, Mrs. Niemish, called me and asked me to make an address of welcome at this testimonial dinner tonight, I gladly accepted the assignment. I was told that I would not be limited in my remarks, nor would there be a time limit.

I am honored by this assignment tonight, because it affords me an opportunity to publicly pay tribute to my esteemed colleague. I have known and worked with Dr. Chipman over the past 24½ years, and during that time, I have come to appreciate his many fine qualities. But I choose not tonight to dwell upon complimentary remarks, for that is the province of the speaker of the evening, but prefer rather to direct your attention to certain aspects of a physician's life which mirrors the sacrifices made by your guest, and which, incidentally, are accepted as a matter-of-fact by the public. Having practiced in this community for nearly 25 years, I feel qualified to discuss these matters and clothe my remarks with a certain aura of authority.

First, I wonder how many of you realize what it means to operate a solo practice on a 24-hour basis, seven days a week? To sleep by the telephone as Dr. Chipman has done for some 45 years? To be constantly interrupted at mealtime by both telephone and doorbell, to spurn the 40-hour week, bankers' hours, bank holidays, paid vacations, and fringe benefits? What, also, does it mean to an individual to put the welfare of his patients first and relegate his own physical welfare, his social life, his associations with his family, and his spiritual life to a secondary position?

I remind you, also, of another facet of a physician's life which is as necessary as all the rest, and which in turn is not seen, and therefore not generally appreciated by the general public. That is the matter of post-graduate education. A large drug company may average a new product from their research departments at the rate of one a week, and there are many drug companies. In the course of a year, many thousands of new products appear on the market. A physician must sift these and apply those which are suitable to the needs of his community. It is said that approximately 75 per cent of the drugs prescribed today were unknown ten years ago. Specialty magazines must be read, new medical books studied, hospital staff meetings attended, as well as medical meetings at stated intervals. It has been stated that a minister could close his Bible and sleep for 100 years, awake and find the Scriptures unchanged, and he could preach as effectively as he did before his long nap. In contrast, a physician who would close his books for a period of a few months would fall woefully behind in the continuing parade of new medications and therapeutic procedures. All of these activities are an added strain on the physician's time and strength.

Your guest has participated in the Golden Age of Medicine. During his years of practice, he has seen and participated in the virtual disappearance of such scourges as typhoid, smallpox, diphtheria, syphilis, whooping cough, and more recently, poliomyelitis. These diseases are now so rare that medical schools have great difficulty in finding a case to present to the students. While not eliminated, the sting has been taken out of pneumonia, tuberculosis, staphylococcal and streptococcal infections, childbearing, and certain types of cancer. Your guest has seen the advent of the sulfonamides, penicillin, a host of mycin drugs, the Salk vaccine, an oral medication for certain types of diabetes, the steroids, heart surgery, the radio isotopes, the application of electronics to therapy, as well as teaching via television.

He has seen the advent of good roads and modern transportation. Gone are the days of parking the car on the highway and walking up to the farmhouses through mud or snow, lugging the necessary equipment and medication—and if at night, counting himself fortunate if the householder carried one of his bags and a lantern to light the way.

Additionally, Dr. Chipman has seen fit to discharge his civic responsibilities to the community as manifest by his past activities in: the Harrington Building and Loan Association, Vice-Presidency of The First National Bank, first President of the Harrington Chamber of Commerce, Director of The Kent and Sussex Fair Association, Speaker of the House to Representatives—to recite but a few.

Let us further take cognizance at this time of the service rendered to Harrington and environs by Dr. Chipman during the grueling 10 years between 1922 and 1933 when he alone gave 24-hour medical service to the community. The fact that he is with us today is a remarkable tribute to the stamina of the human body, and truly his successor will have his tasks appointed in continuing the noble tradition of "service above self" established here in Harrington by Dr. Chipman, and will have, literally and actually, a large pair of shoes to fill. (I am informed that Dr. Chipman wears size 11's!)

I feel that I would be amiss at this gathering if I did not pay a heartfelt tribute to Mrs. Chipman, whose sympathetic understanding, and gracious acceptance of the trials, tribulations, and vicissitudes attendant upon the dual operation of a household and that of a 24-hour telephone operator have been of such esteemable value to Dr. Chipman, and without whom his service to the community would have been impossible.

I take this moment, now, in my capacity as Mayor of the City of Harrington, to warmly welcome all the friends and neighbors and well-wishers who have assembled here in this auditorium tonight to pay their respects to our first citizen. I am proud to lead off with the opening remarks, and I know that the memory of this testimonial dinner will long endure in Dr. and Mrs. Chipman's storehouse of pleasant memories.

And now, back to the Chairman. . . .

*The Chairman then introduced the principal speaker of the evening, Herman C. Brown, Esq. of Dover, one of Dr. Chipman's babies.*

I know of no more happy occasion for me to come back to Harrington than the occasion of tonight. I wish to congratulate, with all the sincerity of which I am capable, every person who has contributed in any manner to this dinner. It is a fete, an occasion and an honor which has been long overdue. Certainly I know no more worthy a person to be honored by his friends than your honored guest this evening, Dr. William Thomas Chipman.

In a way, you know, a testimonial dinner to Dr. Chipman was hardly necessary other than to make it a formal gesture. A testimonial dinner by definition, is one where the friends, associates, colleagues, and contemporaries of the person to be honored gather, and by the presence and by what they say, each gives sincere and heart-felt testimony to the accomplishments and the greatness of the individual to be honored. It is not necessary to come to this very fine dinner tonight to hear such testimony to our honored guest. All that is required is that you be present at any gathering of persons, whether it be formal or informal, in the community in which he has lived and served his fellow men throughout his long career, and the conversation will without fail include plaudits to his accomplishments, to his character, to his personality, to his professional skill and, indeed, to his greatness. If there is anything to the old saying that when a person is discussed in his absence that his ears burn, then most assuredly, our guest's ears must burn constantly. In my lifetime it has never been my pleasure to know a man who is more universally liked, and, in fact, more universally loved, by his community than our guest tonight. History may not record his deeds and his accomplishments, for they have not been performed for the benefit of historians, nor have they been performed for the benefit of publicity or to become renowned. They have been done for the little people, the everyday folk—not the type who write history books, not the type who publish newspapers, and not the type who, because of their great wealth and influence, can cause his name to be recorded in the pages of a formal history. Dr. Chipman's popularity has been with the people who needed him most; the people who at a given instant, were suffering either from their own pain or from the pain of having a loved one in distress. It has been that need that Dr. Chipman has so admirably filled, not only by his professional skill, but far above and beyond that, by his personality, his understanding, and by the tower of strength which he personifies, upon which those persons could lean at the time a shoulder was necessary.

I thought tonight that you might possibly like to know something about Dr. Chipman's early life and background, for despite the fact that you all know him well, you might not have been aware of some of these facts. I thought it might be interesting to all concerned to trace his younger days and, indeed, the movements and the great number of circumstances that finally brought him to our city and to our community.

Born the eighteenth of January, 1891, he was named William Thomas Chipman. Throughout his lifetime he has been known by the members of his profession as Bill. As a boy his friends and school acquaintances referred to him as Tom. In my lifetime, I have never heard him referred to other than as Doc Chipman. This, incidentally, is unusual in itself, since it has been my experience that most doctors find being called "Doc", which seems to be their natural nickname, very distasteful. His family had its roots in England. His grandfather, whose name in fact was Chippenham, came to this country and had three sons. One of these three sons settled in Laurel, in a

community which eventually was called Chipman's Mill. It was here that Dr. Chipman, Tom Chipman, in fact, as he was known then, was born. He lived with his parents in this community and graduated from the Laurel Public School. Following this he entered a private school at Baltimore and remained there for one year. Leaving his private school work, Dr. Chipman enrolled at the Maryland University Medical School in Baltimore and was graduated from there in the year 1912. Following his graduation he interned at Lancaster, Pennsylvania, for approximately one year. Leaving Lancaster, he moved to the Grace Hospital in Detroit, Michigan. He completed his internship at the Grace Hospital in Detroit, and went into private practice there. While he was there he met and married his first wife. After practicing for some short period of time in Detroit, his health broke down and he was advised by the doctors who attended him to leave the city and return to the country. Acting upon this advice, he and his family returned to Delaware, and settled down in the town of Felton, which was the place of his early beginnings in this community. It was there that his oldest child, and son, Berlin, was born in 1914. He built and lived in the house in Felton which is now the Fletcher Nursing Home. His second child, and first daughter, Eloise, was born in Felton in 1916. His wife desired that they should return to Detroit, and his health having improved, in 1919 he and his family left Felton and returned to Detroit. He stayed in Detroit for approximately one year. His mother had died in the year 1915, and in 1920 his father died. Possibly influenced by this, Dr. Chipman returned to Delaware in November of 1920, and for the first time came to the city of Harrington where he resided on Weiner Avenue between what are now the homes of Jacob Messner and Abner Hickman, in a house presently lived in and owned by Herman Longfellow. He commenced practice in the city of Harrington, but his life was not to be as uneventful as he might have hoped practicing in a small town in a rural community, such as this was at that time.

In 1921, in June, his wife died. Following quickly upon the heels of this, the Milford Emergency Hospital first opened its doors in July of 1921, providing for the area for the first time a hospital which meant a great deal to the practice of medicine in this community, and provided, for the first time in the area, operating facilities where Dr. Chipman could practice his art of surgery for which he became so well known in the years to come. In 1922 the only other doctor in Harrington, a Dr. Thompson, left this community, and Dr. Chipman was to be, as Dr. Smith has previously told you this evening, the only doctor in the community from the year 1922 to the year 1933.

In the meantime, however, there had been put into motion another series of events which were, indeed, to have a great effect upon the life of our honored guest. A young lady from Salisbury had graduated from The Memorial Hospital Nursing School in Wilmington in the year 1918, and on the fifteenth day of August of the year 1921, this very capable nurse came to the Milford Emergency Hospital to begin a term of employment there. On the next day, August the sixteenth, as she was preparing to assist another surgeon in an operation at this hospital, this lady was confronted by a very large man wearing a green Norfolk suit, the coat of which he had caught in a pump handle and torn the pocket, who looked very harried and in a great deal of haste, and was, in fact, on his way to Rehoboth to a medical association meeting. To describe it, as she de-

scribed it to me, he was certainly not dressed in a manner to go to a medical association meeting. Dr. Chipman has said in later years that the only reason she was ever interested in him at all, and the only reason that she eventually married him and became the Pearl Chipman that we have known for so many years, was that he looked as though he needed someone to take care of him and see that he was properly dressed to present himself at the various places where his profession and his professional standing required him to go.

Their courtship was a whirlwind affair, as it was described to me by Mrs. Chipman. They courted when their professional duties permitted. Both, as all of you know, were very dedicated persons, and the Milford Hospital in its beginning and its pioneer days required all of the skill, the energy and initiative that two persons such as Doc and Pearl Chipman could give it. Many hardships presented themselves and were overcome by their efforts. For example, Mrs. Chipman was in charge of the operating room, and, at that time, the city of Milford did not have daytime electric power. In order to sterilize instruments, Mrs. Chipman has told me, it was necessary to send the instruments which were thought to be required for the next operation by dumbwaiter down into the kitchen and then returned by dumbwaiter to the operating room after having been sterilized by water heated in the kitchen. However, as time was to prove, they were meant for each other, and they overcame the difficulties which they encountered. Mrs. Chipman at that time had only a room in the nurses home where she was not permitted to entertain dates. Dr. Chipman was still residing in the house on Weiner Avenue, but his first wife's parents were living with him and acting as housekeepers and assisting in the care of his two children. Therefore, there was no place for them to go, and as Doc has often said, the only place they had to court was the Ellendale Swamp. Despite, however, all the adversities which I have outlined, and with which the courtship was faced, it was eventually culminated by marriage. And subsequent thereto, in 1933, a child was born of this marriage, Susan, by name, with whom most of you are familiar.

Because of his gruff exterior, and his direct method of approach, many stories have come into existence in regard to our honored guest. Most of these stories, I am sure, have been heard by most of you. But I have attempted to pick out two or three which I thought were particularly good and particularly interesting, that I might tell you this evening in order that those of you who might not have heard them should have the opportunity to, and in order that those of you who have heard them might enjoy hearing them retold.

One of the stories which I have encountered in my search for folklore in regard to our honored guest goes something like this: It seems that there had come into our community a lady who had been doing practical nursing work in other sections of the country, and upon coming to this community to live was in the process of continuing in her chosen field of endeavor. In the course of this she was attending a patient for whom Dr. Chipman was summoned. Upon arrival, Dr. Chipman burst into the room with his hat and bag in hand in his usual gruff, direct manner, took one quick, percursorial look at the patient, who was lying in bed obviously in great distress, shook out a handful of pills in an envelope, gave his customary "Now, Honey, take one of these just like I wrote on the envelope. You'll be all right. You'll feel better tomorrow," and dashed off in a mad haste. The practical

nurse was somewhat astonished and overcome by what she had seen occur. She dashed out of the room into the hall, chasing after the Doctor, and asked him to stop. When she finally had chased him to the front door, and had caught up with him, she took him to task, telling him that he should have made an examination—he should have taken the temperature of the patient—that he should have made some physical examination—that he shouldn't have diagnosed so quickly—that he should have exercised, in fact, his bedside manner. Having listened to her very patiently for all of some forty-five or sixty seconds, Dr. Chipman, in the course of going out of the door, turned around and said, "Honey, every time you go to make a pan of biscuits, you don't get the cook book down to see how to make them, do you?" And without pause or hesitation, shut the door and continued on his merry way.

Sometime later, however, a young, attractive and very eligible school teacher came to Harrington. She is known to many of you, and she, in fact, still resides in our community. Her name is Leona Dickraker. Shortly after she came to this community, she was taken ill and Dr. Chipman was summoned to her bedside. Apparently deciding to take the advice of the practical nurse, who had previously taken him to task, Dr. Chipman decided that this was a point where his bedside manner might be exercised to its best advantage. Slowing himself down, approaching the situation very calmly, he put down his satchel, laid his hat and coat aside and sat down on the edge of the bed to make a very thorough examination of this particular patient. The unfortunate part of it all was that in the course of sitting down on the edge of the bed, the bed gave way under his weight and broke down, depositing both him and his patient on the floor. It was probably at this point that Dr. Chipman, for all time, gave up any practice of bedside manner, as it is known among physicians.

Probably, however, the most often told story about Dr. Chipman involved a brush with the officers of law enforcement. He has been famous for years for his fast driving and his speed when on a call and his speed to and from the Milford Hospital when his presence was needed for some emergency. In the course of driving, as we all know him to drive, he has had several encounters with the law because of his speed. However, in one particular instance he and another person very well known in Harrington, whose name is Booty Morris, were proceeding down the highway bound for some unknown destination which is not important in the telling of this story. As usual, Doc was exceeding the speed limit. Looking in his rear view mirror, he saw that he was being pursued by a minion of law enforcement. Turning quickly to Booty, and making his usual snap decision, he said, "Booty, throw a fit." Booty, not realizing what was happening, looked around askance. Doc said, "Hurry up, throw a fit. That cop's about to get us." Booty proceeded in accordance with Doc's instructions to throw a fit, going through all sorts of gyrations, pushing himself down on to the floor of the car and putting on quite a display. When the cop drew alongside and commenced to berate Dr. Chipman regarding his driving, Doc's reply was quick, immediate, and gruff, as usual. "My God, man, I'm trying to get this man to the hospital. Can't you see he's having a fit. Get out of my way and let me go." The cop did, and I think it's one of the most fabulous stories in that collection that can be made of stories concerning our honored guest.

Beginning at approximately 1914, when he was living in the town of Felton, and continuing throughout the period from 1920, when he has

resided and practiced in the city of Harrington, the name of "Doc Chipman" has become legend in this community. Those of you who are here tonight, in the main, became acquainted with him when adversity was present in your lives, when you were sick or ill, or when a loved one was in need. He has always been the same. He has always been in a hurry. He'll never be other than gruff, outwardly at least. His efficiency and professional skill have always been of the highest. He has brought a blush to many a young girl's face by his quick remarks and ready wit. He has always been a friend of those who are in need of a friend. But possibly his most important asset is the one that is so intangible that it almost defies description. I refer to his tremendous strength, not only physical strength, but strength of personality and character on which so many persons, and probably most of you, have seen fit to lean in time of need. This includes his ability to carry the burdens of so many other persons, to listen to their trials and their troubles and tribulations; to counsel them as well as to tend them when they are physically ill. His ability, through the use of a very few words, to comfort a person who has just lost a loved one; his ability to inspire confidence in a patient by his very presence; his ability to soothe even a small child who really cannot understand his words, but who senses instinctively that this is the man who will make the hurt better—these are, indeed, the assets required for greatness, and these are a part of the makeup of Dr. Chipman. His ability to walk into a house where a family is gathered because of illness of one of its members, and the confidence which his presence inspires—has ability to walk into a sick room and make a patient feel better before he has set down his bag—these are the attributes of greatness. This is the strength of character, the force of personality and the quiet confidence which oozes from the man. These are also the attributes which would have made him great in whatever his chosen field of endeavor might have been. Had he been a statesman, more likely history would have recorded his acts and deeds, because he would have been a great statesman. Had he been a general, he would have been a great general; again the world would have heard of

Tom Chipman. Instead he was a doctor. Not only was he a doctor, but he chose to practice in this rural community where transportation was difficult, and where his size was an asset to the pushing of a Model T out of the mud. And also where communications were limited, and where his prowess and ability could be recognized only by those whom he served. That is the reason that it is time that his friends, his patients, his associates should gather here tonight to pay tribute to this man who has given so much of his life, his energies, his strength and his skill to this community, to its individual members and to those of you who are gathered here tonight. The fact that he has so chosen to lead a more anonymous life in a rural community only magnifies, in my estimation, the greatness of the man of which I have already spoken.

Let it suffice to say that a speaker could spend the entire evening extolling his virtues without running the risk of being repetitious. Many individual cases and situations could be pointed out. You could be told of his many accomplishments in civic and political fields, which the other speakers have covered this evening. I would like to conclude what I have to say about Dr. Chipman in this manner. I would like to say that in 1920, by a simple decision to return to the city of Harrington to establish a practice, it was the world's loss and Harrington's gain. It was the addition to this community of a man who has given more of himself to the community than anyone who has resided here in this same period or, in fact, in any period. I think that though it be an ordinary phrase and could not be considered eloquent, that there is only one way which, speaking for the community, I can express our appreciation to you, Dr. Chipman. I can only say, "Thank you." I do say this from the bottom of my heart, and in so doing, I speak for all those assembled here. I would like to say further that the standing ovation and the applause which you are about to hear is neither for this speaker nor for any alleged eloquence of his delivery. The applause which you are about to hear, Dr. Chipman, is the method by which these persons, these friends of yours, gathered here, are also saying, "Thank you, Dr. Chipman."

## **What is the Difference Between THE WORLD MEDICAL ASSOCIATION and THE WORLD HEALTH ORGANIZATION?**

### **THE WORLD MEDICAL ASSOCIATION**

1. WMA is an organization of national medical associations. The unit of membership is the most representative national medical association in each country. It is completely non-governmental. It is not part of the U.N. It is a voluntary organization.
2. WMA represents the practicing medical profession.
3. WMA was organized in 1947 by AMA representatives and Western European medical leaders. Purpose was to exchange medical knowledge, to protect the freedom of medicine, and promote world peace.
4. Each member association sends two delegates, two alternate delegates and observers to the General Assemblies — the supreme policy making body of WMA.
5. The executive body of WMA is the Council. This meets twice a year and comprises 11 members elected from the Assembly and the President, President-Elect and Treasurer.
6. WMA is supported by members' dues and contributions and the annual budget is about \$165,000.
7. American physicians and allied corporations interested in the work of WMA

are organized as the United States Committee of The World Medical Association.

### **THE WORLD HEALTH ORGANIZATION**

1. WHO is an intergovernmental health agency. The members are the governments that accept the nine principles upon which WHO is founded.
2. WHO represents governments in their public health and medical activities.
3. WHO is the result of proposal of U.N. in 1945 to create a specialized agency to deal with all matters related to health on a world-wide scale.
4. Each member government sends three delegates, chosen preferably from the national health administration of the government, to the annual World Health Assembly.
5. The Executive Board of WHO is the executive body and consists of 18 members elected to represent 18 member governments.
6. WHO is supported by dues allocated by the U.N. scale and the budget for 1958 is \$13,000,000.
7. American citizens interested in the work of WHO are organized as the Citizens' Committee for the World Health Organization.

## THE AMERICAN CONGRESS OF PHYSICAL MEDICINE AND REHABILITATION

The 36th annual scientific and clinical session of the American Congress of Physical Medicine and Rehabilitation will be held August 24-29, 1958 inclusive, at The Bellevue Stratford Hotel, Philadelphia.

Scientific and clinical sessions will be given August 25, 26, 27, 28, and 29. All sessions will be open to members of the medical profession in good standing with the American Medical Association.

Full information may be obtained by writing to the Executive Secretary, Dorothea C. Augustin, American Congress of Physical Medicine and Rehabilitation, 30 North Michigan Avenue, Chicago 2, Illinois.

Professional people who are called upon to investigate and report on fluoridation, will now find a package library of authoritative reference material available to them at the library at the Academy of Medicine

in Wilmington, or will be able to borrow the material, on a "loan basis", from the State Department of Health.

Among other items, the package library presently contains reports from the National Research Council, and technical papers on the "Medical Aspects of Excessive Fluoride in a Water Supply", the "Non-dental Physiological Effects of Trace Quantities of Fluorine", and "Urinary Fluoride Levels Associated with Use of Fluoridated Waters".

The "loan" copy of the package library can be borrowed for a period of one week, by writing to

Miss Margaret H. Jeffreys, Director  
Division of Oral Hygiene  
State Board of Health  
Dover, Delaware

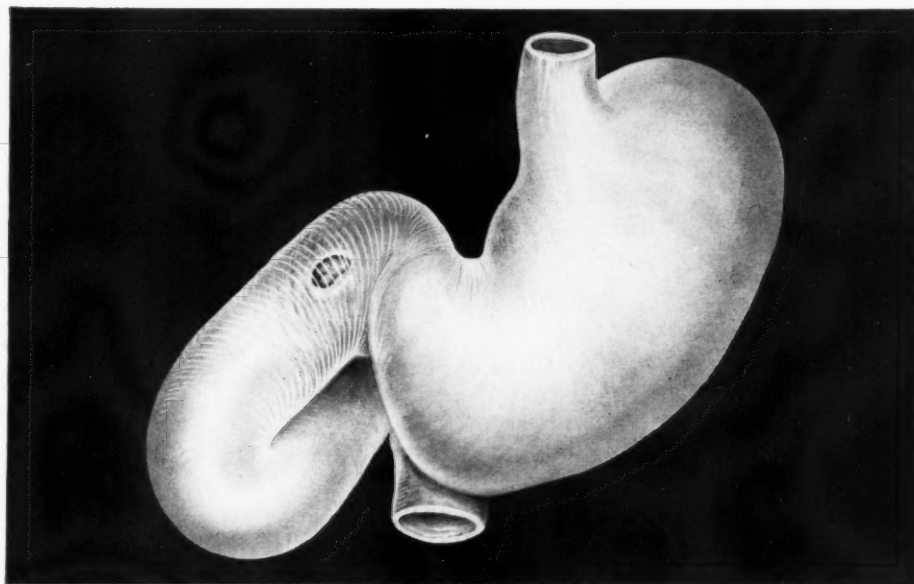


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\*Lichstein, J.; Morehouse, M. G., and Osmon, K. L.: Pro-Banthine in the Treatment of Peptic Ulcer. A Clinical Evaluation with Gastric Secretory, Motility and Gastroscopic Studies. Report of 60 Cases, Am. J. M. Sc. 232:156 (Aug.) 1956.

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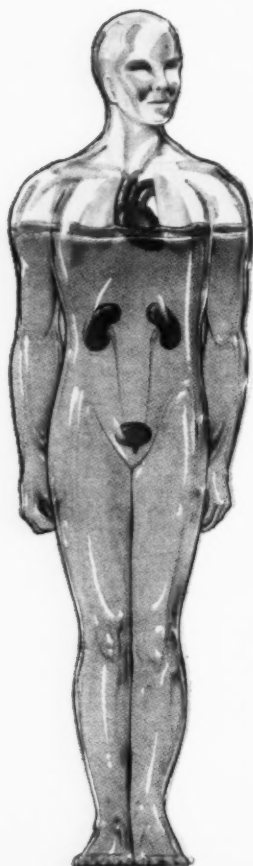
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## 3 ADJUST DOSAGE OF ALL MEDICATION

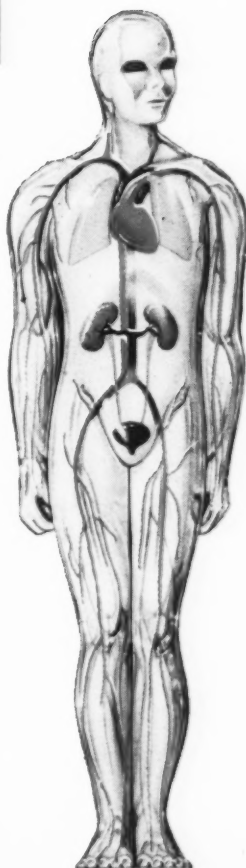
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Riseman, J. E. F., Altman, G. E., and Koretsky, S.:  
Nitroglycerin and Other Nitrites in the Treatment of  
Angina Pectoris, *Circulation* (Jan.) 1958.

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May we send more complete information and bibliography.

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\*Tests in a series of 25 patients show that there is "a definite and distinct lowering [of both volume of secretions and of free hydrochloric acid] in the majority of patients. . . . No patients had shown any increase in gastric secretions following administration of the drug."<sup>1</sup>

Now you have 4 advantages when you calm ulcer patients with ATARAX:

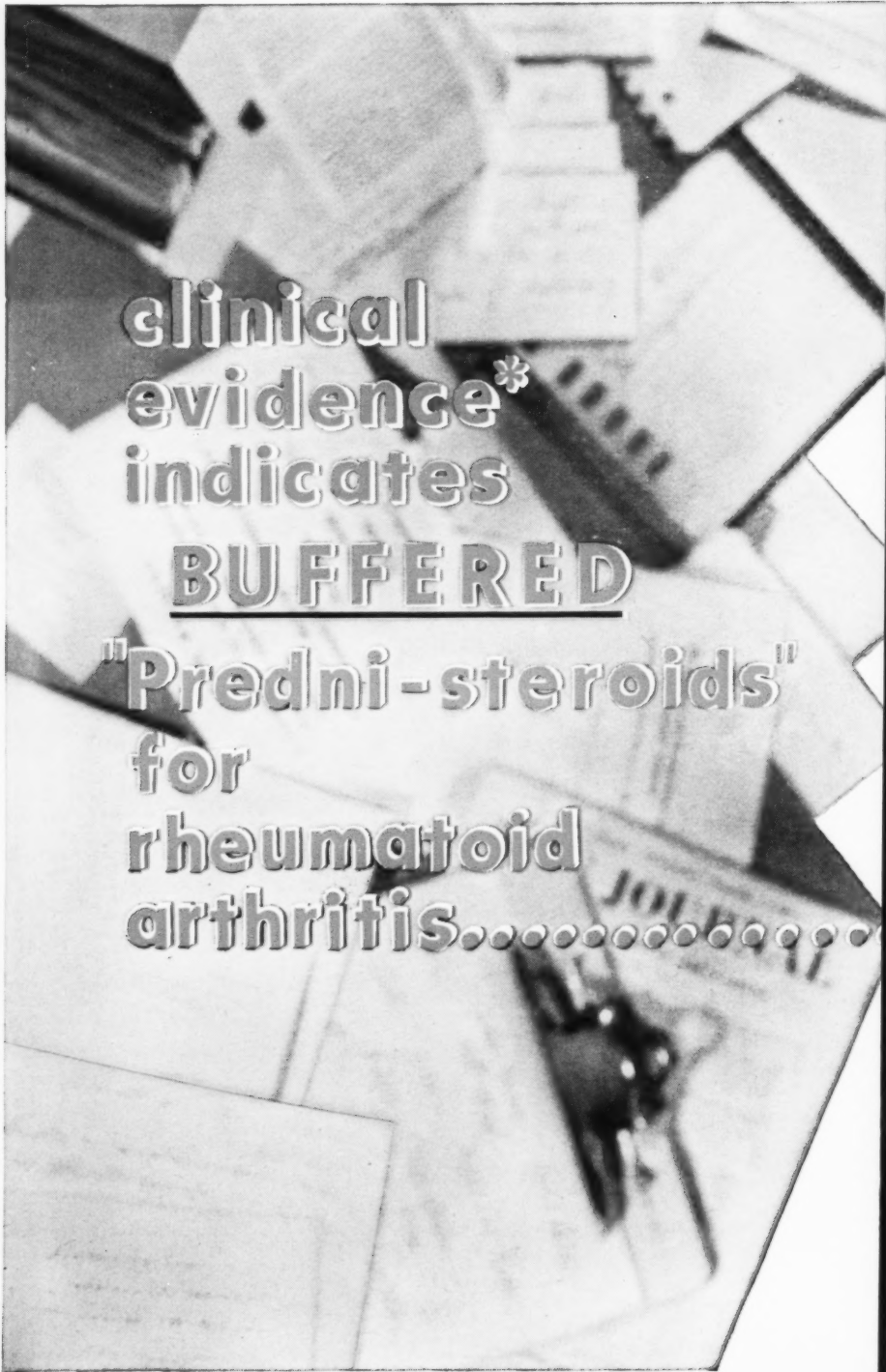
1. ATARAX suppresses gastric secretions; others commonly increase acidity.
2. ATARAX is "the safest of the mild tranquilizers."<sup>2</sup> (No parkinsonian effect or blood dyscrasias ever reported.)
3. It is effective in 9 of every 10 tense and anxious patients.
4. Five dosage forms give you maximum flexibility.

**supplied:** 10, 25 and 100 mg. tablets, bottles of 100. Syrup, pint bottles. Parenteral Solution, 10 cc. multiple-dose vials.

**references:** 1. Strub, I. H.: Personal communication. 2. Ayd, F. J., Jr.: presented at Ohio Assembly of General Practice, 7th Annual Scientific Assembly, Columbus, September 18-19, 1957.

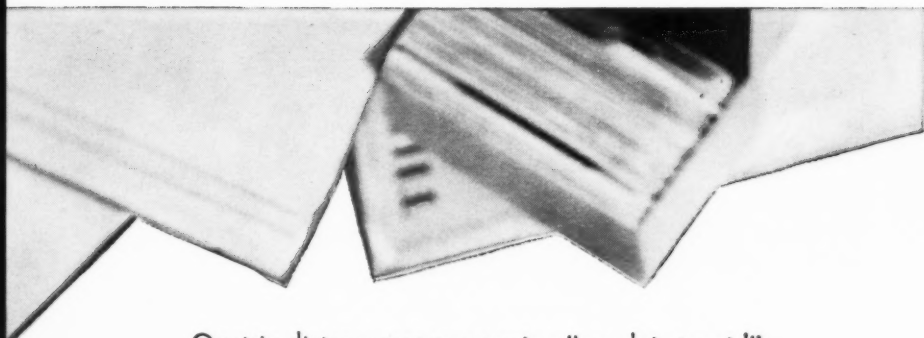


New York 17, New York  
Division, Chas. Pfizer & Co., Inc.

A black and white photograph of a desk with papers, a pen, and a stapler. The text is overlaid on the image.

**clinical  
evidence\*  
indicates  
BUFFERED**

**"Predni-steroids"  
for  
rheumatoid  
arthritis.....**



Gastric distress accompanying "predni-steroid" therapy is a definite clinical problem — well documented in a growing body of literature.

\*"In view of the beneficial responses observed when antacids and bland diets were used concomitantly with prednisone and prednisolone, we feel that these measures should be employed prophylactically to offset any gastrointestinal side effects."—Dordick, J. R., *et al.*: N. Y. State J. Med. 57:2049 (June 15) 1957.

\*"It is our growing conviction that all patients receiving oral steroids should take each dose after food or with adequate buffering with aluminum or magnesium hydroxide preparations."—Sigler, J. W. and Ensign, D. C.: J. Kentucky State M. A. 54:771 (Sept.) 1956.

\*"The apparent high incidence of this serious [gastric] side effect in patients receiving prednisone or prednisolone suggests the advisability of routine co-administration of an aluminum hydroxide gel."—Bollet, A. J. and Bunim, J. J.: J. A. M. A. 158:159 (June 11) 1955.

One way to make sure that patients receive full benefits of "predni-steroid" therapy plus positive protection against gastric distress is by prescribing CO-DELTRA or CO-HYDELTRA.

**Co-Deltra**  
PREDNISONE BUFFERED

multiple compressed tablets

provide all the benefits  
of "Predni-steroid" therapy—  
plus positive antacid protection  
against gastric distress

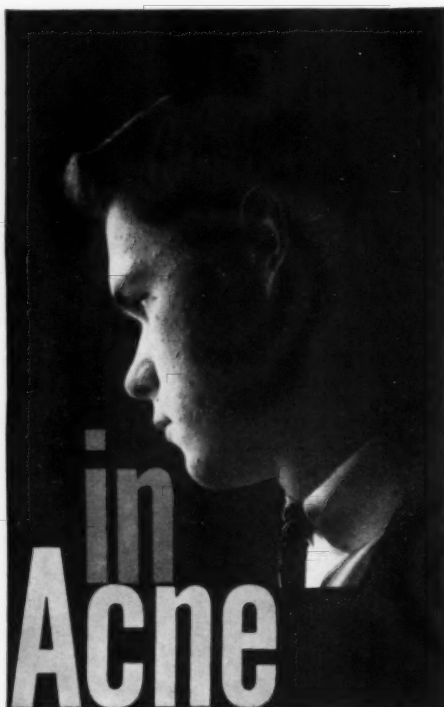
**Co-Hydeltra**  
PREDNISOLONE BUFFERED



2.5 mg. or 5.0 mg. of prednisone or prednisolone, plus 300 mg. of dried aluminum hydroxide gel and 50 mg. magnesium trisilicate, in bottles of 30, 100, 500.

MERCK SHARP & DOHME Division of MERCK & CO., INC., Philadelphia 1, Pa.





"No patient failed to improve."

*pHisoHex washing added to standard treatment in acne produced results that "...far excelled... results with the many measures usually advocated."*<sup>1</sup>

pHisoHex maintains normal skin pH, cleans and degerms better than soap. In acne, it removes oil and virtually all skin bacteria *without scrubbing*.

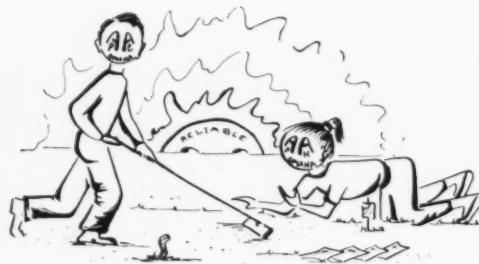
For best results—four to six washings a day with pHisoHex will keep the acne area "surgically" clean.

1. Hodges, F. T.: *GP* 14:86, Nov., 1956.

# pHisoHex®

Sudsing  
nonalkaline  
antibacterial  
detergent—  
nonirritating,  
hypoallergenic.  
Contains 3%  
hexachlorophene.

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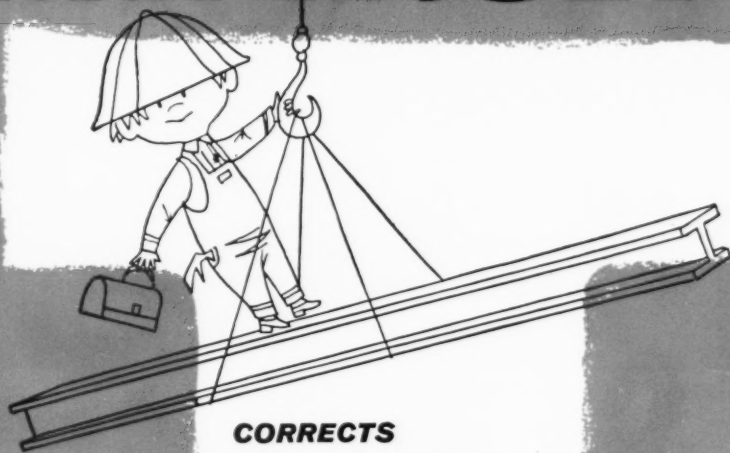
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IRON DEFICIENCY  
AS IT  
STIMULATES  
APPETITE**

**DELICIOUS CHERRY FLAVOR  
DESIGNED TO APPEAL TO  
BOTH CHILDREN AND ADULTS**

**PARTICULARLY FOR CHILDREN**

Supplies essential Iron as ferric pyrophosphate, highly stable, well-tolerated, readily absorbed; essential vitamins B<sub>1</sub>, B<sub>6</sub> and B<sub>12</sub>, established as appetite stimulants; essential L-Lysine for greater protein economy in the pediatric diet.



## **INCREMIN Syrup**

**FORMULA:** Each teaspoonful (5 cc.) contains:

L-Lysine HCl	300 mg.
Ferric Pyrophosphate (Soluble)	250 mg.
Iron (as Ferric Pyrophosphate)	30 mg.
Vitamin B <sub>12</sub> Crystalline	25 mcgm.
Thiamine Mononitrate (B <sub>1</sub> )	10 mg.
Pyridoxine HCl (B <sub>6</sub> )	5 mg.
Alcohol	0.75%

Average dosage is 1 teaspoonful daily.  
Available in bottles of 4 fl. oz.

\*REG. U. S. PAT. OFF.

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OF DERMATOSES...**  
(Regardless of Previous Refractoriness)

**Confirmed by  
an impressive and  
growing body of published  
clinical investigations** \*

**TARCORTIN** CREAM  
Hydrocortisone 0.5% and Special Coal Tar Extract 5%  
(TARBONIS®) in a greaseless, stainless vanishing cream base.

**NEO-TARCORTIN** OINTMENT  
Hydrocortisone 0.5%, Neomycin 0.35% (as Sulfate) and Special  
Coal Tar Extract 5% (TARBONIS) in an ointment base.

ATOPIC DERMATITIS • ECZEMAS • SEBORRHEA • ANOGENITAL PRURITUS • DERMATITIS VENERATA • PRURIASIS

\* J.A.M.A. 166:158, 1958; Welsh, A.L. and Ede, M.  
"...prompt remissions of...acute phases."  
with TARCORTIN

**R&C REED & CARRICK** / Jersey City 6, New Jersey

\* 1. Clyman, S. G.: Postgrad. Med. 21:309, 1957.  
2. Bleiberg, J.: J. M. Soc. New Jersey 53:37, 1956.  
3. Abrams, B. E. and Shaw, C.: Clin. Med. 3:839, 1956.  
4. Welsh, A. L., and Ede, M.: Ohio State M. J. 50:837, 1954.  
5. Bleiberg, J.: Am. Practitioner 8:1404, 1957.

## Doctors, too, like "Premarin"



The reasons are fairly simple. Doctors like "Premarin," in the first place, because it really relieves the symptoms of the menopause. It doesn't just mask them — it replaces what the patient lacks — natural estrogen.

Furthermore, if the patient is suffering from headache, insomnia, and arthritic-like symptoms before the menopause

and even after, "Premarin" takes care of that, too.

Women, of course, like "Premarin," too, because it quickly relieves their symptoms and gives them a "sense of well-being."

**"PREMARIN"**  
conjugated estrogens (equine)



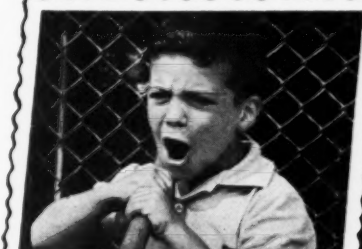
Ayerst Laboratories • New York 16, New York • Montreal, Canada

# MY DAD— HE HURT HIS BACK REAL BAD

"It happened  
at work  
while he  
was putting  
oil in  
something"



"He told  
Mom his  
shoulder  
felt like  
it was on  
fire"



"He couldn't  
swing a bat  
without  
hurting"



"But Doctor  
gave him  
some nice  
pills--and  
the pain  
went away  
fast"



"Dad said  
we'd play  
ball again  
tomorrow  
when he  
comes home"



AND THE PAIN  
WENT AWAY FAST

## FOR PAIN Percodan® TABLETS

(Salts of Dihydrohydroxycodone  
and Homatropine, plus APC)

**ACTS FASTER...**  
usually within 5-15 minutes

**LASTS LONGER...**  
usually for 6 hours or more

**MORE THOROUGH RELIEF...**  
permits uninterrupted sleep through the night

**RARELY CONSTIPATES...**  
excellent for chronic or bedridden patients

## and now... **NEW** Percodan- Demi

### VERSATILE

New "demi" strength permits dosage flexibility to meet each patient's specific needs. PERCODAN-DEMI provides the PERCODAN formula with one-half the amount of salts of dihydrohydroxycodone and homatropine.

**AVERAGE ADULT DOSE:** 1 tablet every 6 hours. May be habit-forming. Available through all pharmacies.

Each PERCODAN® Tablet contains 4.50 mg. dihydrohydroxycodone hydrochloride, 0.38 mg. dihydrohydroxycodone terephthalate, 0.38 mg. homatropine terephthalate, 224 mg. acetylsalicylic acid, 160 mg. phenacetin, and 32 mg. caffeine.

*Literature? Write*

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release from pain and inflammation

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**IN ARTHRITIS**

salicylate benefits with  
minimal salicylate drawbacks

**Rapid and prolonged relief** — with less intolerance. The analgesic and specific anti-inflammatory action of BUFFERIN helps reduce pain and joint edema—comfortably. BUFFERIN caused no gastric distress in 70 per cent of hospitalized arthritics with proved intolerance to aspirin. (Arthritics are at least 3 to 10 times as intolerant to straight aspirin as the general population.<sup>1</sup>)

**No sodium accumulation.** Because BUFFERIN is sodium free, massive dosage for prolonged periods will not cause sodium accumulation or edema, even in cardiovascular cases.

Each sodium-free BUFFERIN tablet contains acetylsalicylic acid, 5 grains, and the antacids magnesium carbonate and aluminum glycinate.

Reference: 1. J.A.M.A. 158:386 (June 4) 1955.

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**Bristol-Myers Company**  
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Relieve moderate or severe pain

Reduce fever

Alleviate the general malaise of  
upper respiratory infections

**'TABLOID'**

**'EMPIRIN'  
COMPOUND<sup>®</sup>  
WITH  
CODEINE  
PHOSPHATE<sup>\*</sup>**

maximum codeine analgesia/optimum antipyretic action

\*Subject to Federal Narcotic Regulations



**BURROUGHS WELLCOME & CO. (U.S.A.) INC., Tuckahoe, New York**

**Symbols  
OF  
PROVEN  
PAIN  
RELIEF**



**gr. 1**



**gr. ½**



**gr. ¼**



**gr. ⅛**

# Formulas for dependable relief...

...from moderate to severe pain complicated by tension, anxiety and restlessness.

## 'CODEMPIRAL' No. 3\*



Codeine Phosphate	gr. 1/2
Phenobarbital	gr. 1/4
Acetophenetidin	gr. 2 1/2
Aspirin (Acetylsalicylic Acid)	gr. 3 1/2

## 'CODEMPIRAL' No. 2\*



Codeine Phosphate	gr. 1/4
Phenobarbital	gr. 1/4
Acetophenetidin	gr. 2 1/2
Aspirin (Acetylsalicylic Acid)	gr. 3 1/2

...from pain of muscle and joint origin, simple headache, neuralgia, and the symptoms of the common cold.

'TABLOID'

## 'EMPIRIN' COMPOUND®



Acetophenetidin	gr. 2 1/2
Aspirin (Acetylsalicylic Acid)	gr. 3 1/2
Caffeine	gr. 1/2

...from mild pain complicated by tension and restlessness.

## 'EMPIRAL'®

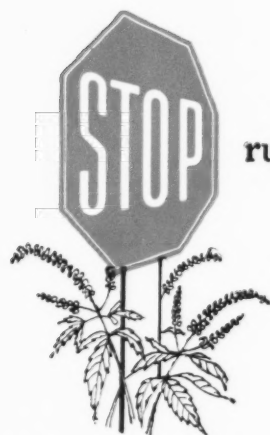


Phenobarbital	gr. 1/4
Acetophenetidin	gr. 2 1/2
Aspirin (Acetylsalicylic Acid)	gr. 3 1/2

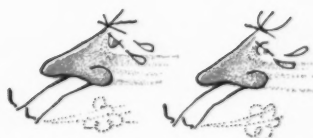
\*Subject to Federal Narcotic Regulations



BURROUGHS WELLCOME & CO. (U.S.A.) INC., Tuckahoe, New York



running noses ...



caused by pollen allergies

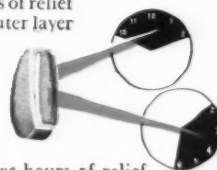
**TRIAMINIC** stops rhinorrhea, congestion and other distressing symptoms of summer allergies, including hay fever. Running nose, watery eyes and sneezing are best relieved by antihistamine *plus* decongestant action—systemically—with **TRIAMINIC**.

This new approach frequently succeeds where less complete therapy has failed. It is not enough merely to use histamine antagonists; ideally, therapy must be aimed also at the congestion of the nasal mucosa. Triaminic provides such effective combined therapy in a single timed-release tablet.

**TRIAMINIC** brings relief in minutes—lasts for hours. Running noses stop, congested noses open—and stay open for 6 to 8 hours.

*Triaminic provides around-the-clock freedom from allergic congestion with just one tablet t.i.d. because of the special timed-release design.*

*first—3 to 4 hours of relief from the outer layer*



*then—3 to 4 more hours of relief from the inner core*

**Dosage:** One tablet in the morning, mid-afternoon and at bedtime. In postnasal drip, one tablet at bedtime is usually sufficient.

#### Each timed-release **TRIAMINIC** Tablet contains:

Phenylpropanolamine HCl .....	50 mg.
Pheniramine maleate .....	25 mg.
Pyrilamine maleate .....	25 mg.

#### **TRIAMINIC FOR THE PEDIATRIC PATIENT**

**TRIAMINIC Juvelets\***, providing easy-to-swallow half-dosages for the 6- to 12-year-old child, with the timed-release construction for prolonged relief.

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**TRIAMINIC Syrup**, for those children and adults who prefer a liquid medication. Each 5 ml. teaspoonful is equivalent to  $\frac{1}{4}$  Triaminic Tablet or  $\frac{1}{2}$  Triaminic Juvelet.

# Triaminic<sup>®</sup>

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## THE ENCOURAGEMENT OF PROGRESS

The American Cancer Society's annual Spring Crusade is the climax of its year-round attack on cancer through research, professional and lay education, and service to the stricken. A study of the cancer scoreboard indicates that steady progress is being made. More and more lives are being saved. Progress encourages more progress.

Earlier diagnosis, new methods of treatment and a greater public awareness have contributed to this progress. It is often said that the life of the cancer patient is in the hands of the first physician he consults. The Society, therefore, conducts a broad professional education program, making available to doctors, through literature, films, exhibits, and other materials, information on the latest advances in detection, diagnosis and treatment.

As the Society aids the doctor, so does its large corps of volunteers aid the cancer patient with dressings, transportation, home care, medication and a host of other vitally needed services.

For the past two years, the theme of the Society's annual Crusade has been "Fight Cancer with a Checkup and a Check." That Americans everywhere are learning the value of the annual health checkup in the fight against cancer, is evidenced by the fact that doctors report they are now seeing more cancer in its earliest stages than ever before.

That American men and women have a personal stake in the program of the American Cancer Society is demonstrated by the public's generous support of the Crusade. This year the goal is \$30,000,000 and we are confident that our people will meet the challenge... will "fight cancer with a checkup and a check" in the encouragement of further progress.

Lowell T. Coggeshall, M.D., *President*  
*American Cancer Society*



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CLINICAL BRIEFS FOR MODERN PRACTICE*Which plasma proteins may be hazardous in renal disease?*

The globulins. They are more easily precipitated to form casts with blockage of renal tubules. The greater the damage to the glomeruli, the greater the proportion of urinary globulin to albumin and subsequent tubular impairment.

Source—Hoffman, W. S.: The Biochemistry of Clinical Medicine, Chicago, The Year Book Publishers, Inc., 1954, p. 233.

*colorimetric "dip-and-read" test  
for proteinuria***ALBUSTIX<sup>®</sup>**  
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just dip...

...and read in mg. %

for tablet testing—ALBUTEST<sup>®</sup> Reagent Tablets detect proteinuria with one drop of urine.



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For prophylaxis: 'Compazine' Spansule† capsules provide all-day or all-night relief of anxiety with a single oral dose. *Also available:* Tablets, Ampuls, Multiple dose vials, Syrup and Suppositories.

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